

The American Journal of Surgery

EDITOR: THURSTON SCOTT WELTON, M.D., F.A.C.S., NEW YORK

IN THIS ISSUE:

THIRTEEN ARTICLES ON

GOITER and Other Diseases of the Thyroid Gland

By S. D. VAN METER, RICHARD B. CATTELL, CHARLES GORDON HEYD, ADA MARIE SMITH, LEO LOEB, ROBERT OLESEN, WALTER M. SIMPSON, VICTOR E. CHESKY, J. R. YUNG, GULBRAND LUNDE, JOHAN HOLST, RALPH T. RICHARDS, WILLIAM O. THOMPSON, PHEBE K. THOMPSON, W. H. PRIOLEAU.

ALSO ARTICLES ON

SPINAL ANESTHESIA, TORSION OF THE NORMAL
FALLOPIAN TUBE, TRAUMATIC SURGERY,
HERNIA, CANCER, ELECTRO-SURGERY
etc., etc.

IN ALL 33 ORIGINAL CONTRIBUTIONS

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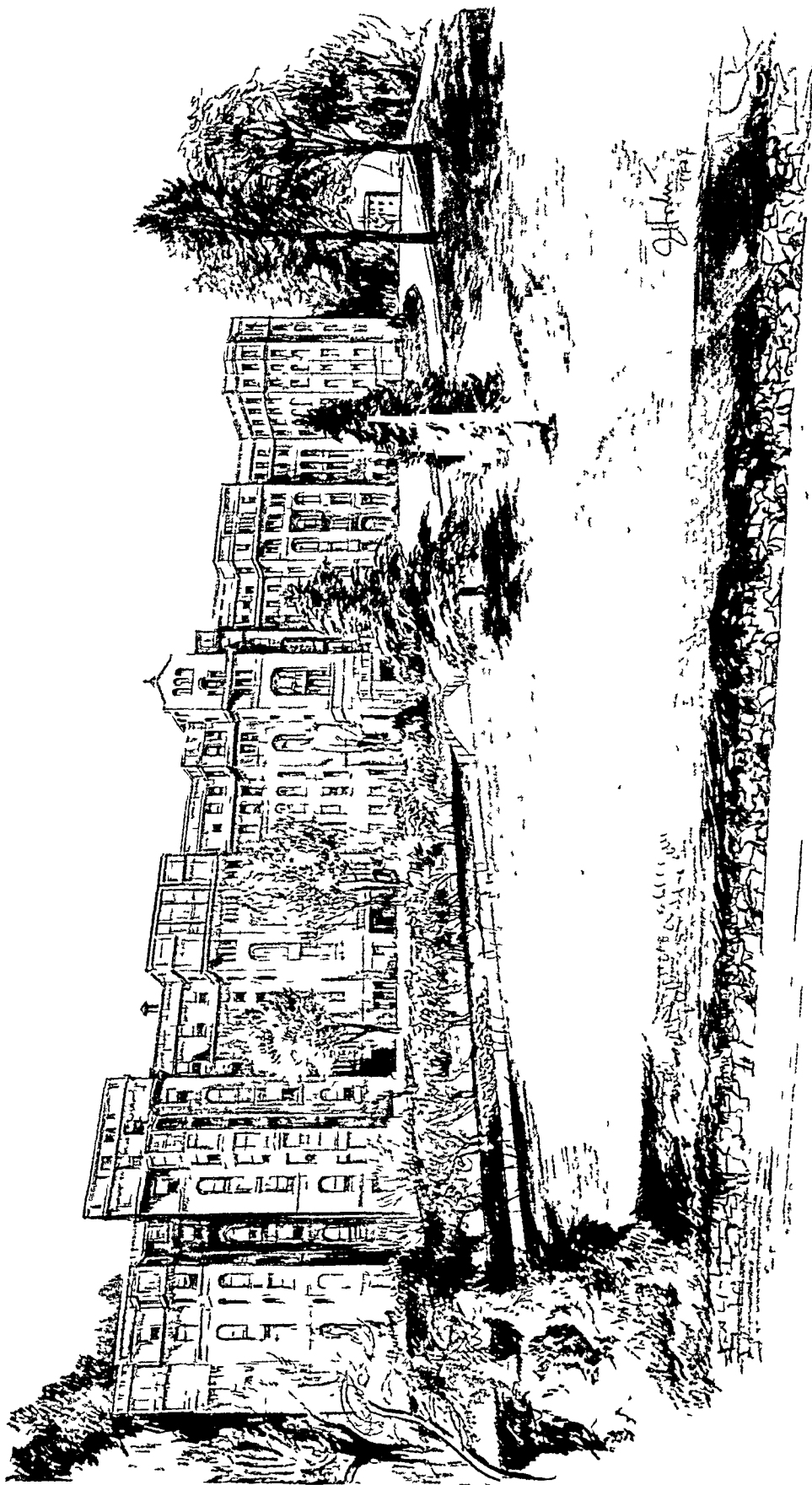
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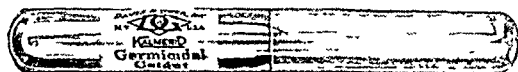
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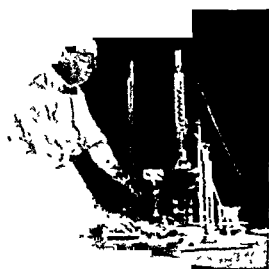


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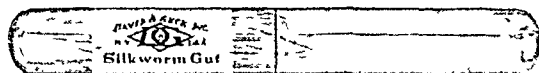
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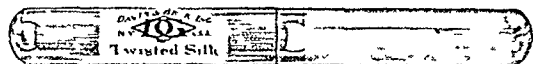


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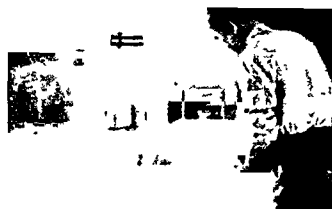


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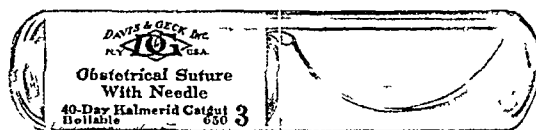
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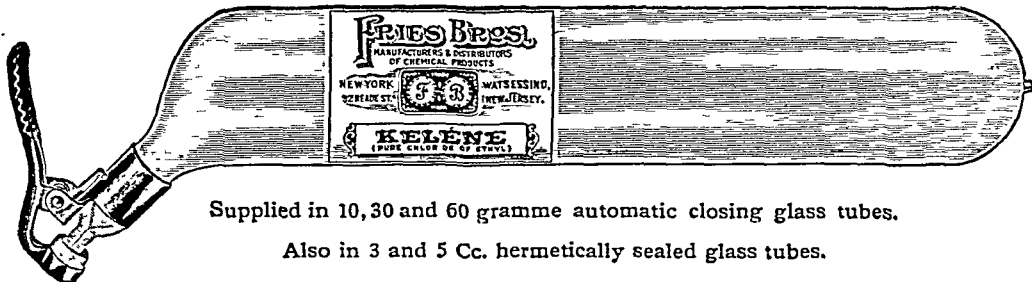
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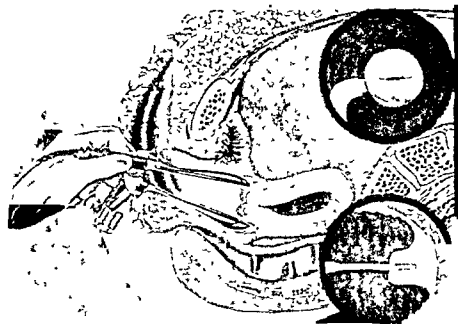
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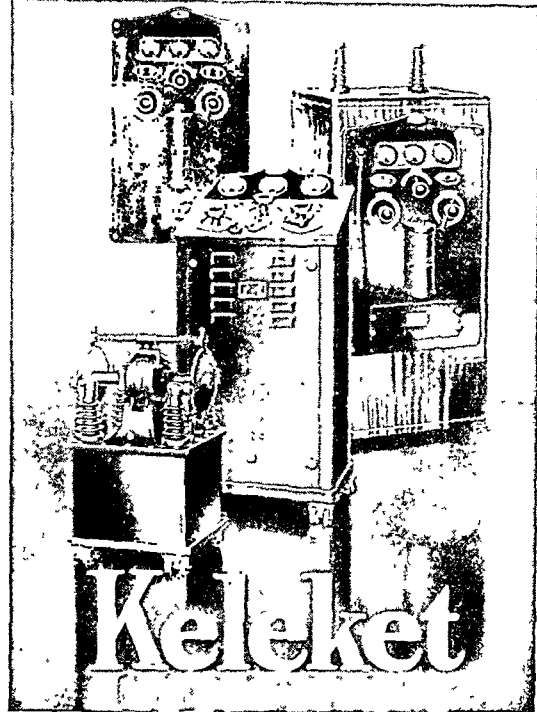
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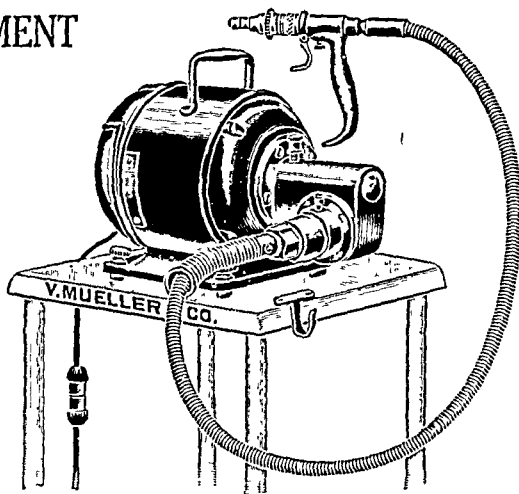
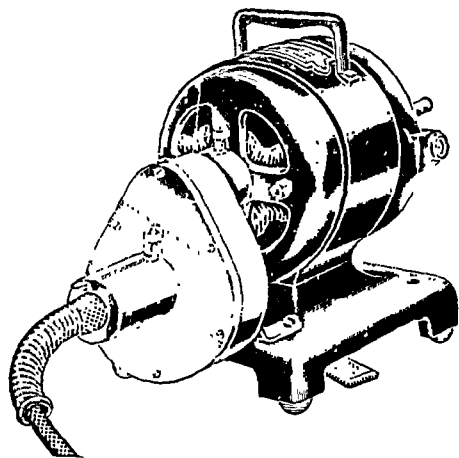
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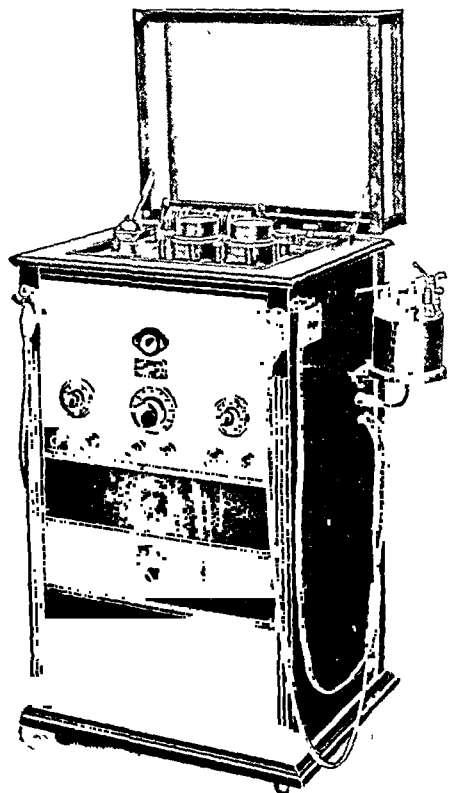
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No. 1

ORIGIN, AIMS & POLICY OF THE AMERICAN ASSOCIATION FOR THE STUDY OF GOITER*

S. D. VAN METER, M.D.

DENVER

THE unavoidable, everchanging personnel of the attending membership of this Association renders it impossible for many of those present at this meeting to know much about the origin and aims of our Association. Consequently I came to the conclusion that nothing would be more appropriate or serve better the interests of the organization than for me to address you upon "The Origin, Aims and Policy of the American Association for the Study of Goiter."

The origin of this Association is to be found in the fact that for years men interested in the goiter problem were dissatisfied with the scant attention paid to the subject in the existing medical organizations.

Six years ago, at Bloomington, Illinois, on the initiative of Dr. E. P. Sloan, the American Association for the Study of Goiter was organized with the definite object of creating a society devoted wholly to the study of goiter and its associated problems, a society that would bring together each year men who would present the best that had been thought, said and done relative to this important, well-known, but poorly understood disease. To Dr. Sloan is due the honor of starting the organization. In addition we must

credit him with the conception of its democratic plan and policy, as well as an untiring zeal in aiding its development and progress. From its very beginning the founders were convinced that the Association, if expected to succeed, should be a representative body. To accomplish this we felt that the active membership should be distributed equally among the several states. We believed that a liberal policy in regard to membership should be adopted if we were to create and maintain widespread interest in the study of the goiter problem.

We proceeded on the principle, that if you cannot think a thing out yourself, you should get as many people as you can to thinking on the problem, that by so doing, somebody may find facts that have eluded you, and through them come to the solution. Who thinks a matter out is of no importance whatsoever. The important thing is that the problem should be solved.

This was the theory upon which the originators of our Association based their hopes of progress and ultimate success in the solution of the unsolved phases of goiter.

To encourage widespread interest in our programs and work, attending membership was extended to all doctors in

* Presidential Address read at the Annual Meeting of the American Association for the Study of Goiter, Dayton, March 25-27, 1929.

good standing in their respective state and provincial medical societies, upon presentation of their credentials and payment of a small registration fee. Such members have all the privileges of active members save those of holding office and participating in the business affairs of the Association.

The limitation of active membership was for no other purpose than to insure fair trial of the plan and policy adopted, which would by uniform distribution of the active membership throughout the continent make the Association truly representative, and furthermore not allow it to become sectional in character. Some of the states are still not represented in our Council, but a sufficient number of active memberships have been reserved for them when they conclude to join in the active management of the Association.

A survey of those states not represented in the active membership shows they are states where there is not much goiter. In our opinion, however, there is sufficient goiter in every section of the country to warrant the deep interest of the profession of those sections even where the disease is not prevalent, in the progress and the general affairs of this Association.

If there are attending members present at this meeting from any of those states not represented in our active membership, I beg of them to carry this message to the profession of their respective states: The American Association for the Study of Goiter is desirous of having active members from every state in the Union and from every province in the Dominion of Canada.

Particularly are we anxious to have members from every state and province who are willing, enthusiastic workers, and who are in accord with our aims and policy.

That the policy of our Association is good and founded on the right principles, we think is conclusively proved by the record it has made in the short time of

its existence. It has established a forum where all subjects pertaining to goiter may be presented and discussed by any member of the profession in good standing in his local medical society. That it has stimulated study and interest in the goiter problem is well shown by the rapid improvement in each successive annual scientific program. Now since it has been thoroughly established and organized, no one need be afraid of its failure, as some did in the beginning.

Aside from the stimulus to study and research such an organization creates, and through which we should expect discoveries that will tell us many things about goiter we fain would know, there are many things our Association can and should do that will be most helpful to every one interested in goiter. I will call your attention to only two of these. The first is that of concerted effort to reform goiter nomenclature, which, as you all know, is in many respects misleading. Even the definition in the most modern medical dictionary (Gould) of the term *goiter* is ridiculous: *A tumor of the thyroid gland.*

A disease of the thyroid gland characterized by disordered function, with or without enlargement of the organ, would better convey the idea of the general conception of the entity we call goiter. The numerous names in use for the type of the disease commonly known as *exophthalmic goiter* is another example which illustrates the necessity for simplification and clarification of goiter nomenclature. The mere fact that *exophthalmos* is a striking feature of this type of the disease in less than 50 per cent of cases, does not warrant the use of the term *exophthalmic*.

The constant features of hyperplasia and toxic symptoms in this type, however, justify the present tendency of the profession to call it *toxic hyperplastic goiter*, and should serve as a guide to those desirous of bringing about reform. Certain it is that the present nomenclature and the numerous lengthy and involved classi-

fications of the disease are responsible for much confusion and misunderstanding, which could be avoided by sensible reform.

The elimination of the ordinary infections and primary neoplasms of the thyroid, which should not be classed as goiter, permits adoption of the simple classification of:

1. Colloid
2. Nodular
3. Hyperplastic

Adoption of this short classification of goiter would go a long way toward bringing about a clearer and more workable grasp by the general profession of the goiter problem. I can conceive of no type of the disease that will not fall under some subheading or combination of these three types.

The other thing that this Association can do, and which would be of inestimable assistance in the study of goiter, is the establishment of a council that will, from time to time, publish in abstract the consensus of opinion on important questions relative to the disease. The scheme would entail considerable labor on the part of the council or committee in charge, but the great good that it would accomplish in an educational way would be full recompense for the effort expended.

In conclusion allow me again to express my sincere appreciation of the honor of having served this Association as its presiding officer for the year, and assure you that as long as I am able to aid in furthering its noble aims, my services are at your command. With your high ideals you cannot help but make continued progress and you should be the means by which the mystery of the etiology of goiter may be solved. This must come before we can expect progress in the prevention or rational treatment of the disease which, sad to admit, at the present time is but little superior to that in vogue in China centuries before the dawn of the Christian Era. We should not allow our pride in the progress made in the management of the advanced stages of the disease to obscure the importance of finding its primary cause. Without this essential cornerstone our foundation is imperfect, any superimposed structure unstable, and sure to fall like the proverbial house of cards.

Consequently, no greater honor lies hidden in the future than that awaiting him who wins the crown of laurel, so justly due the discoverer of the basic cause of goiter. By that achievement, in all probability, one more scourge of the human race will be conquered.



PARATHYROID TRANSPLANTATION

A REPORT OF AUTOGRAFTS OF PARATHYROID GLANDS REMOVED DURING THYROIDECTOMY*

RICHARD B. CATTELL, M.D.

BOSTON

TRANSPLANTATION of the parathyroid glands has been limited, with few exceptions, to experimental work in animals. Reports of such investigations followed soon after the identification of the parathyroids by Sandström in 1880. The most important early work was the establishment by Gley that postoperative tetany was due to the removal of these bodies alone. An attempt to cure the tetany produced by the excision of these glands by transplantation logically followed. Kocher, Eiselsberg, Enderlen, Payr, and others transplanted them together with portions of thyroid tissue with indifferent success. Christiani and Ferrari were able to show normally growing transplanted parathyroid tissue in cats for more than five years. Biedl reported 2 successful autotransplantations in dogs. Both Pool and Pepere found such transplantations unsuccessful in controlling postoperative tetany in animals. The latter showed that the grafts would control the tetany until absorption took place. Leischner proved the functional capacity of such grafts in rats by excising them; this procedure was followed by death from tetany.

The most important work in this field is the well-known work of Halsted. He was successful in 60 per cent of his autotransplantations in a large series of dogs. He recognized that his failures were due to the fact that the animal did not require this transplanted tissue. Thus in order to obtain successful transplantations it was necessary to create a deficiency by injury or removal of the remaining parathyroids at the time of the transfer. This was the reason for the indifferent results of the previous investigators. Halsted further

showed that one gland was sufficient to protect the animal from tetany. In fact, he maintained one dog for several months with a transplant measuring $\frac{1}{4}$ by $\frac{1}{2}$ mm. Death followed its removal. He was unsuccessful in isotransplants in 38 dogs.

The technique of transplantation has varied considerably in these experiments. Kocher placed them in the bone marrow of the tibia. Others transplanted them in the spleen, intestine, muscle and thyroid. Halsted placed them under the rectus abdominis muscle where they could be identified from the peritoneal side.

Transplantation of human parathyroid glands for the control of postoperative tetany has been carried out a few times. Eiselsberg transplanted one removed during another goiter operation to a woman suffering with severe chronic tetany which followed total thyroidectomy. This apparently was successful, although it should be noted that she had been able to get along with the disease for several years before transplantation. Garré reported success in a case of chronic tetany with transplantation into the tibia. Pool and Turnure transplanted a gland from a young man to another of their patients with tetany following an operation for recurrent goiter. Tetany occurred on the fourth postoperative day; transplantation was done on the sixth day, and there were no further symptoms after the seventh day. This transplantation was done under the rectus muscle. It is quite likely that this was a temporary tetany, since relief followed too soon after transplantation for the graft to be vascularized. It is possible it acted as a temporary benefit until complete absorption took place, but most likely

* Read at the Annual Meeting of the American Association for the Study of Goiter, Dayton, March 25-27, 1929.

it had nothing to do with the course of the disease. Brown reported a very severe case of tetany following two hemithyroidectomies. He transplanted three parathyroids taken shortly after death from a man who succumbed with chronic nephritis. He reported complete relief.

In the Lahey Clinic 2 human transplantations have been done; in each case the parathyroid was taken from another patient.

CASE I. A girl of twenty-one had a subtotal thyroidectomy for exophthalmic goiter. At operation two parathyroid-like bodies were found on the excised gland and transplanted by the usual technique. Neither was a parathyroid, but both were shown to be portions of thyroid tissue by microscopic section. On the day following operation she developed severe tetany with a blood calcium below 7 mg. She was sent home on calcium and parathormone, but returned in four weeks with a blood calcium of 6.2 mg. Transplantation of two parathyroid-like bodies was done six weeks after operation by Dr. Lahey. Neither was examined histologically, since it was felt it might further injure them. This patient was not relieved. She died five and one-half months after thyroidectomy.

CASE II. This patient had a subtotal thyroidectomy in the Clinic in 1920, with a recurrence of symptoms after four years. A large remnant was removed in 1926. No parathyroids were found on the specimen. Four days later she developed severe clinical tetany with a blood calcium of 5.6 mg. With calcium therapy she was relatively free from symptoms and maintained a blood calcium of 6 to 7 mg. She received parathormone daily with improvement. A parathyroid was transplanted six months after the onset of tetany by Dr. Mason. Her condition remained the same for ten days, following which she needed less parathormone and calcium. It was not felt this could be attributed to the transplantation.

The functional test as carried out in experimental animals by excision of the transplant obviously cannot be applied to human cases. If a patient with severe clinical tetany and low blood calcium is relieved by such a graft and acquires a

normal calcium, then one must concede that it is successful in spite of the failure of all experimental isografts of a similar nature. The actual success of each of these reported cases seems open to question on these grounds.

In 1924, the author, while observing the effect of iodine on the structure and chemistry of the thyroid gland in exophthalmic goiter, carefully sectioned all portions of 200 such glands removed at operation. All nodules suspected of being parathyroids were examined microscopically and their position on the gland noted. Fourteen parathyroids were found, an incidence of 7 per cent of the operative specimens. These parathyroids were found in all positions, although usually at the junction of the middle and lower thirds of the lobe on the lateral surface. When here they were usually in relation to the middle thyroid vein or inferior thyroid artery. One was found deeply imbedded in the gland, others on the anterior surface or near the superior pole.

Today all successful thyroidectomies are modified to protect the parathyroids. Numerous plans have been presented for their preservation, and I do not wish to discuss the technical steps by which this can be done. The technique of thyroidectomy as carried out in the Lahey Clinic exposes the posterolateral surface of the lobes by division of the prethyroid muscles. Parathyroids had been occasionally recognized and preserved at operation before 1924. In 1925, Dr. F. H. Lahey suggested careful examination of the excised tissue at the time of operation under aseptic conditions, with immediate transplantation of parathyroids when found. He reported his experience with this procedure that year. Since that time, this has been a routine part of all our goiter operations.

Parathyroid glands can be recognized in more than 10 per cent of thyroid operations. As one's experience increases, the number identified at the time of operation rises. This affords greater protection

against possible tetany, since one can modify the resection to preserve them with their blood supply intact. They are easiest to see in the exophthalmic type of gland or in any goiter in which the thyroid keeps its general outline in spite of its increase in size. When an adenoma occupies the lobe, it may completely distort the lateral surface and make identification difficult. This is particularly true in substernal goiters.

Besides position, the small nodules of thyroid tissue seen in adenomatous goiter may be readily confused. At times there may be similar projections from the surface in exophthalmic goiter. Lymph glands are occasionally found in the same region but can usually be differentiated. In an occasional instance we have confused them with accessory portions of thymus tissue or tabs of fat. A positive identification can only be made by a microscopic section. We have had verification, by this means, of bodies thought to be parathyroids over one hundred times, and feel that we can now identify many of them at operation. Certainly it should be emphasized that when any doubt exists, such bodies should be preserved.

Identification of the upper parathyroids at operation is infrequent. We have found them in relation to the anterior branch of the superior thyroid vessels and preserved them. In order to remove sufficient thyroid tissue in exophthalmic goiter, it is not practical to leave parathyroids in any position except in relation to the two arteries. Frequently, on division of the middle thyroid vein, a parathyroid is visualized. It lies in a loose mesh of areolar tissue (erroneously called the capsule) and can be demonstrated to have a separate blood supply. These parathyroids are oval or fusiform bodies, usually slightly movable, varying in color from brownish yellow to brownish red. Their size is variable, but is usually about 6 by 3 by 2 mm.

It will be appreciated that with the recognition of a greater number of para-

thyroids during operation, there will be fewer removed, and fewer found on the excised tissue. The same means of identification are used in the search made on the excised portion of the thyroid gland. The technique of transplantation has been fully reported by Dr. Lahey. A thin section is removed from each for microscopical verification and the rest is placed in a pocket made in the sternomastoid muscle. This should be a dry field to prevent the graft from being included in a blood clot.

THE INCIDENCE OF TRANSPLANTATION

In 1925, transplantation was carried out 32 times. During the time up to March, 1929, transplantation had been done 340 times in 332 patients. Of these, 125 were shown to be actual parathyroids by microscopical examination. Thus 33.8 per cent of all nodules identified at operation on the excised tissue as possible parathyroids were proved to be such. Approximately 80 per cent of these were taken from exophthalmic goiter patients. During this four-year period (1925-1928) 4148 thyroid operations were done. It will be seen from these figures that approximately 8 per cent of all thyroid operations during this period have had transplantation of parathyroids as a part of the operative procedure. If one adds to this the 10 per cent of cases where parathyroids have been visualized during operation and left undisturbed, it will be seen that identification of parathyroids during operation is a rather common incident when search is made. This is contrary to the usual surgical experience reported in the literature.

INCIDENCE OF TETANY

Tetany has been a rather rare complication of thyroid operations in the Lahey clinic. There have been no recognized cases with clinical tetany during the past two years. We have had 11 patients with definite tetany in 6682 goiter operations, an incidence of 0.2 per cent. Of these 11 patients, 8 recovered completely after a brief interval and were symptom-free

without treatment. The remaining 3 patients had severe permanent tetany. In 2 parathyroid isograft transplantation was done without relief as described earlier.

During the past two years we have frequently taken blood calcium determinations when certain sensory disturbances were noticed after operation. In none of these were the Trousseau or Chvostek signs positive and in each the blood was within normal limits. During the same time we have followed the same operative technique and have routinely transplanted all parathyroids found.

Of the 11 patients who had postoperative tetany, 3 had had the transplantation carried out at operation.

The first was reported earlier in the paper, with two parathyroid-like bodies removed and transplanted, but both proved to be thyroid tissue only. She developed tetany on the following day, was unimproved by an isograft from another patient, and died in five and one-half months. A second patient had two parathyroids, proved by sections, removed at a first-stage hemithyroidectomy. There was no evidence of tetany. Six weeks later the second stage hemithyroidectomy was done; no parathyroids were seen on the removal specimen. Five days later she developed tetany with a blood calcium of 7.1 mg. This patient has been under our continuous observation for two and one-half years. She takes large amounts of calcium by mouth and required parathormone for several months after operation. Her blood calcium drops to 6.8 mg. when treatment is discontinued. The third patient had a subtotal thyroidectomy for multiple colloid adenomatous goiter with a parathyroid, positively identified, being transplanted. Forty-eight hours later she developed severe tetany with a positive Chvostek and Trousseau signs, and cramps of hands and feet. The blood calcium at this time was 6.6 mg. She was given calcium chloride intravenously with immediate and complete relief. Her blood calcium on successive days on calcium therapy were 8.5, 7.9, 7.1, 8.0, and 8.5 mg. Nine days after operation and seven days after the onset of tetany, her blood calcium was 9.6 mg. and she had no signs of tetany with calcium therapy discontinued.

These 3 patients should be considered carefully. The first was obviously unsuccessful since the bodies transplanted were not parathyroids. The second had two proved parathyroids transplanted at the first of two operations. The left side of the gland was not touched at the first operation. No tetany resulted. But tetany did result after the second operation. The transplants did not "take" because there was no deficiency at the first operation and they were absorbed before the second operation six weeks later. This situation is exactly the same as Halsted's unsuccessful experiments. The third patient developed tetany two days after operation and with temporary calcium therapy during the period of vascularization of the graft, the transplanted parathyroid was able to carry on the normal function of the parathyroids. The conditions represented in this patient satisfy all the requirements of a successful transplantation with the exception of excision of the graft which is followed by tetany.

The incidence of tetany has dropped noticeably since we have adopted transplantation routinely. This lowered incidence is, we believe, coincident with transplantation, but not wholly due to it. For undoubtedly the wide exposure of the thyroid, particularly at the common locations of the parathyroids, has led indirectly to a better protection for them.

We have been further led to these conclusions by experimental work by Dr. R. L. Mason and the author, and I should like to make a brief preliminary report of these experiments. We have attempted to reproduce the conditions of transplantation as carried out in our patients. All the parathyroids have been removed from dogs by parathyroidectomy. Acute fatal tetany with a low blood calcium follows in thirty-six to seventy-two hours. In others we have removed all parathyroids and transplanted one or more in the sternohyoid or sternomastoid muscles. Under these conditions we were surprised to see fatal tetany again occur. We then found that

under these conditions where transplantation has been done, if the animal is given calcium and an active parathyroid extract for a brief interval, the animal will be preserved and the transplant will maintain a normal blood calcium without signs of tetany. These experiments are still in progress and will be reported later in detail. One dog was kept for three months without signs of tetany after the initial postoperative period. Myxedema developed. Excision of the transplant with its surrounded muscles after this three months' period was followed by a lowered blood calcium, and death from tetany in forty-eight hours. Epithelial cells were found in the area of the excised transplant in the microscopic sections. Thus chemical, structural, and functional proof of a successful transplant is demonstrated in dogs under identical conditions as can occur in our goiter operations where parathyroids have been removed. It must be appreciated, however, that it was necessary to maintain these dogs by parathyroid extract for an appreciable time after operation in order for them to survive and be successful.

This situation was reduplicated in but 1 patient, and a successful relief followed which can possibly be attributed to it. It may be, also, as Pepere first suggested, that in many patients the transplanted glands may be absorbed over a few days' period and exert a useful function in possible transient and mild tetany. We have not felt justified in excising the area of the transplant in human cases, since it might lead to tetany. Dr. Clute has excised one such transplant, but found it to be actively growing thyroid tissue. This

corresponded to the section taken at the time of transplantation.

CONCLUSION

In conclusion, it will be seen that the experimental transplantation of parathyroids can be successfully carried out in dogs where a deficiency is created and the animal is maintained over a necessary interval by calcium and parathyroid therapy. No verified isotransplantation (that is, from one animal to another) has been successful. On this basis, the relief of severe postoperative tetany following goiter operations by transplantation of a gland from another patient is improbable and there is little chance of success with this method. This has been borne out by our experience in 2 patients. We feel encouraged in continuing the transplantation of all parathyroids found on the excised thyroid gland during operation and believe from our experimental work and 1 successful human case that if a definite parathyroid deficiency does result, a transplantation of this type will be adequate to prevent permanent tetany. This seems to be the best protection against tetany when one or more parathyroids have been removed.

We would urge all surgeons performing operations for goiter to familiarize themselves with the usual position and appearance of parathyroid glands; to examine the excised tissue for possible removed glands; and to carry out their transplantation when found as has been described. Finally, it should be recognized that the most important factor in the prevention of postoperative parathyroid tetany is their preservation by a suitable operative technique.



RECTAL ANESTHESIA (GWATHMEY) IN EXOPHTHALMIC GOITER*

CHARLES GORDON HEYD, M.D., F.A.C.S., AND ADA MARIE SMITH, R.N.

NEW YORK

IN the treatment of any surgical condition of the thyroid gland there are embraced three well-defined periods: (1) the preparation of the patient, (2) the operation, and (3) the postoperative treatment.

PREOPERATIVE ROUTINE

In thyroid conditions not associated with toxicosis a prolonged preoperative treatment is not required, nor is the giving of iodine preoperatively indicated. In this report we are concerned with rectal anesthesia in the severe types of thyrotoxicosis, but more particularly the toxicosis of the so-called Graves' disease type. The preoperative treatment embraces the following:

1. *Diet.* High caloric, forced feeding: small meals at frequent intervals.

2. *Rest.* Absolute rest and quiet in bed, occasionally allowing the patient up one hour a day in order to preserve muscle tone.

3. *Fluids.* Administration of fluids by mouth and rectum: by rectum in the form of Murphy drip, made up of tap water 500 c.c. and glucose 50 gms., every eight hours. For the twenty-four hours immediately preceding operation 2 c.c. of Lugol's solution are added to each 500 c.c. of water.

4. *Medication.* Luminal in $\frac{1}{4}$ to $\frac{1}{2}$ grain doses, three or four times a day.

5. *Nursing.* Special nurse an invaluable ally, particularly if of the right type and experience.

6. *Ice Bag.* Ice bag alternate hours to heart and thyroid.

7. *Iodine.* Lugol's solution in doses of 1 to 2 c.c. in a full glass of water, one-half hour after meals for three to five days before operation, and the last twenty-four hours before operation 2 c.c. per 500 c.c. of water by rectum.

8. *Transfusion.* Occasionally, although rarely, indicated: not more than 500 c.c. of blood by direct method.

9. *Duodenal or Levine Tube.* If vomiting and nausea are present a very valuable aid in giving fluids by means of the Murphy drip applied to the tube which passes either through the mouth or nose.

10. *Anesthesia.* Of great importance is the selection of the type and the administration of anesthesia. For the last two years we have employed rectal anesthesia, as devised by Gwathmey, for operative work in the desperate cases of Graves' disease. Occasionally rectal anesthesia is employed for hyperthyroidism of adenoma. In all other goiters, with infrequent exceptions, ethylene gas anesthesia is used.

RECTAL ANESTHESIA

With rectal anesthesia it is essential to have the very active cooperation of the anesthetist, one who has been thoroughly trained and combines patience and technical efficiency. With rectal anesthesia the patient does not immediately absorb a large amount of ether upon its introduction into the rectum; the absorption takes place gradually and the etherization is smooth and continuous because the rate of ether evaporation is constant. It is essential that the calculated amount of ether as computed by age, weight and general condition of the patient be given all at the one time in order to produce the proper degree of analgesia or anesthesia. According to Gwathmey, the originator of the method, the absorption from the rectum is at the rate of $1\frac{1}{2}$ to 2 ounces per hour and likewise the elimination takes place at approximately the same rate, $1\frac{1}{2}$ to 2 ounces per hour.

Recently we have employed a prelimi-

* Read at the Annual Meeting of the American Association for the Study of Goiter, Dayton, March 25-27, 1929.

nary try-out in that forty-eight hours before the scheduled operation the patient is not given breakfast and 1 ounce of ether and 3 ounces of olive oil are introduced into the rectum. The bed is then screened, the room darkened, quietness enforced and the patient has from three to four hours of apparently normal sleep, from which she is readily awakened but has a surprising amount of analgesia. This preliminary test is sometimes of advantage in the unusually toxic case where the slightest rectal irritability or burning might be translated into severe pain. It has the added psychic advantage that when the patient emerges from this induced sleep and finds she has not been operated upon she is very apt to ascribe it to a therapeutic procedure for her condition. When the next rectal introduction is made the patient is not suspicious that any unusual procedure is directed against her.

The exact technique of the Gwathmey rectal anesthesia as carried out at the New York Post-Graduate Hospital is as follows:

A definite hour for the beginning of the operation is determined upon and it is of the utmost importance that the operating room and surgeon be ready to operate on schedule time. The night before operation the patient takes pulvis glycyrrhizae compositus ℥ii to ℥iii at 8 P.M. The operation is scheduled to begin at 2 P.M. the following day. At 7 A.M. the morning of the day of operation a soap suds enema is given, followed in one hour by a colonic irrigation of clear warm water until the return flow is clear. This usually requires from 6 to 10 gallons of water given through a bi-way rectal tube. The enema and colonic irrigation should be completed by 9 A.M. At 9:15 A.M. morphine grain $\frac{1}{8}$, $\frac{1}{6}$ or $\frac{1}{4}$ is given, depending upon the body weight, height and age of the patient. The patient is kept in bed and absolute quiet is insisted upon from this time on. At 12 o'clock noon, chloretone, grain xv, ether ℥ii , olive oil ℥ss , is given as a retention enema

through a tube that has been inserted into the rectum a distance of 8 to 10 inches. The rectal tube is allowed to remain in situ and is clamped to prevent reflow. At 12:50 P.M. a second dose of morphine, grain $\frac{1}{8}$, $\frac{1}{6}$ or $\frac{1}{4}$ is given hypodermatically, the dosage to be determined as already indicated by weight, height and age of the patient, together with an estimation of the reaction obtained by the previous dose of morphine as given at 9:15 A.M. With this morphine is also given $\frac{1}{150}$ grain of atropine hypodermatically. The patient is then placed in a left Sims' position and at 1 P.M. the following mixture is prepared and given per rectum: Olive oil ℥ii , ether ℥iv , and paraldehyd ℥i . One ounce of this mixture for each 20 pounds of body weight is introduced through a funnel connected with the tube that has already been placed in the rectum.

Some patients complain of cramps, burning sensation and a desire to evacuate the bowels. To relieve this condition it is advisable to stop the flow of the solution and, by lowering the tube, allow the flatus to escape without losing any of the mixture. When the required amount has been given the tube is again clamped and allowed to remain in the rectum throughout the operation.

If the patient has not become narcotized by the time the mixture is put in, the anesthetist allows the patient to rebreathe, preferably with a Bennet inhaler. This must be accomplished without the knowledge of the patient that anything has touched the face. The rebreathing prevents any loss of the anesthetic through the expired air. Once the operation is started further anesthesia is unnecessary. The length of time that patients have been held under this type of anesthesia has varied greatly, but in 1 case an anesthesia of one hour and forty-five minutes was obtained which should give ample time for almost any type of operative work.

Rectal anesthesia requires a very carefully detailed plan properly coordinated for its success. In about 4 out of 5 cases

we are able to do the thyroid operation without the patient having any knowledge as to when it is to be performed or any remembrance of the operation until she awakened after its completion.

POSTOPERATIVE TREATMENT FOR THE FIRST TWENTY-FOUR HOURS

Upon the completion of the operation the tube, which has remained in the rectum, is unclamped and the mixture, if any, is allowed to drain off. This is measured and the amount withdrawn is deducted from the original amount. As soon as the patient is returned to bed a colonic irrigation of cold water is given immediately, followed by a retention enema of olive oil ℥iv to ℥vi .

Morphine, grain $\frac{1}{6}$, hypodermatically every four to six hours for pain or restlessness.

Fluids. Water by mouth as soon as consciousness returns. If a Levine tube has been introduced through the nose, or a duodenal tube through the mouth, the Murphy drip is given continuously through this tube. Proctoclysis, 500 c.c. every eight hours, consisting of tap water 500 c.c., glucose 50 gm., Lugol's solution 2 c.c.

Position: Fowler or sitting position.

Medication: Digitalis, if indicated. We do not digitalize our patients before operation unless there is definite cardiac

disability. For fibrillation quinidine grain iii every four to six hours.

ADVANTAGES OF RECTAL ANESTHESIA

1. Complete absence of psychic impressions.
2. Preservation of the normal color during anesthesia.
3. Maintenance of normal blood pressure, pulse and respiration rhythm during anesthesia.
4. Less bleeding.
5. Freedom from etherizing paraphernalia about head and face.
6. Absence of complications.
7. Anesthesia maintained during operation and postoperative analgesia continued four to six hours afterward.
8. Safety and lessened mortality.

SUMMARY

Goiter with Hyperthyroidism (130 cases)

Hyperthyroidism	
Graves' disease type.....	89
Adenoma type.....	37
Carcinoma of thyroid.....	4

Basal Metabolism

Highest.....	127+
Lowest.....	22+
Average.....	45+

Postoperative Complications Referable to Anesthesia

Rectal pain.....	3
Bloody stool ninth day.....	1
Cramps in abdomen.....	2
Pneumonia.....	1
Cough.....	2
Vomiting.....	5
Mortality.....	1



THE EFFECT OF POTASSIUM IODIDE, THYROID & ANTERIOR PITUITARY SUBSTANCE ON THE THYROID GLAND OF THE GUINEA PIG*

LEO LOEB, M.D.

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IN the center of the present discussion of structure and function of the thyroid gland, under normal and abnormal conditions, is probably the effect of iodine on this organ. In general, the view represented by Marine is accepted: Iodine diminishes the activity of the gland; it causes the production of hard colloid, low epithelium; it acts similarly to administration of thyroid substance.

This assumption rests on the following statements:

(a) Iodine largely prevents the occurrence of endemic goiter, if given to young children, and it may, in certain cases, reduce early goiter.

(b) In administering iodine to dogs, the colloid becomes hard, and the epithelium compressed and flat. According to Marine hyperplastic goiters are transformed in dogs into colloid, resting goiters.

(c) According to Marine, iodine prevents the occurrence of compensatory hypertrophy, which occurs after removal of a part of the gland; it may be recalled here that the structure of the thyroid in compensatory hypertrophy is the same as that found in a typical, very pronounced case of Graves' disease.

(d) If iodine is administered to patients suffering from Graves' disease, it reduces, at least temporarily, the basal metabolism and it reduces also other symptoms of the disease. As far as structure is concerned it has been maintained that it causes the acini to become extended with solid, hard colloid, the epithelium to become flattened and the connective tissue to be increased in amount.

Contrary to this view, Hellwig finds that, under these conditions, the hypertrophic character of the gland persists and that merely the consistency of the colloid increases. Furthermore, there can be no doubt that administration of iodine, especially in cases of non-toxic diffuse or adenomatous colloid goiter, may transform the simple into toxic goiter and may produce even the symptoms of Graves' disease.

We have then to deal with two kinds of evidence: under certain conditions iodine apparently leads to a diminution, whereas under other conditions it leads to a stimulation of the activity of the thyroid gland. As far as experiments in animals are concerned, they all seemed to point, so far, to a lowering of the thyroid activity under the influence of iodine leading to a quiescent state in this organ.

It may, therefore, be of interest to report on a series of investigations in the guinea pig, which apparently are contrary to the almost generally accepted view that iodine diminishes the activity of the thyroid gland, and which point in the other direction, namely, that it may stimulate it. And here it may be recalled that, as far as structure is concerned, the stimulated condition of the thyroid is recognized by an increase in the size of the acinar epithelium and by a softening of the colloid, which may be followed by an almost complete absorption of this substance; in some cases the liquefied colloid may be retained in the acini and exert pressure on the acinar epithelium, at the same time distending the acini. In addition the stimulated condition of the

* From the Dept. of Pathology, Washington Univ. School of Medicine, St. Louis. Read at the Annual Meeting of the American Association for the Study of Goiter, Dayton, March 25-27, 1929.

thyroid gland may find expression in an increased number of phagocytes, which help to dissolve the colloid, and in an increased number of mitoses. These various changes often go hand in hand, but apparently this is not always the case; there may be instances in which there is marked hypertrophy without a corresponding high number of mitoses and, on the other hand, there may be a very pronounced increase in the number of mitoses without a corresponding marked hypertrophy of the gland.

The investigations, the results of which I wish to summarize here, were begun about ten years ago and they concerned, at first, a study of compensatory hypertrophy of the thyroid gland in the guinea pig. In general, we found that the degree of hypertrophy observed under apparently similar conditions varies extremely in intensity in different individuals and in a considerable number of cases hypertrophy may be absent. However, here we are interested mainly in the action of certain substances on compensatory hypertrophy. Contrary to expectation, we observed that this condition is not prevented by the administration of potassium iodide. Under the influence of this substance we found many pronounced cases of compensatory hypertrophy and, on comparing statistically the thyroids in the control and in the potassium iodide series, both comprising a very large number of animals, the larger number of hypertrophied glands was observed in those animals to which iodine had been administered. We used as a criterion for hypertrophy, in these cases, the increase in size of the epithelium, irregular shape of the acini and the softening of the colloid. We found the softening of the colloid very much more pronounced in the guinea pigs administered potassium iodide than in the control animals. This softening occurred in patches, which alternated with areas in which no softening occurred. In many cases the softened colloid distended the acini in spots and thus a variegated appearance of the thyroid

was produced which frequently permitted us to diagnose the previous administrations of iodine to such animals. There was generally a larger number of mitoses found in hypertrophied thyroids than in normal glands, but comparative counts in controls and in animals given potassium iodide have not yet been made.

The determination of the relative frequency of hypertrophy in controls and in animals given potassium iodide is rendered more difficult through the interference of a number of variable factors in this process. Thus, I found that compensatory hypertrophy is less liable to occur in the warm season of the year and furthermore I observed that the general state of health of the animal, as indicated by change in weight during the course of the experiment, influences markedly the degree of hypertrophy; in guinea pigs that lose in weight, hypertrophy is, on the average, much less pronounced than in animals which show a considerable gain in weight. From the evidence on hand, I would be inclined to predict that, in cases which, during the period of iodine administration, show a marked gain in weight, the compensatory hypertrophy will, on the average, be found to be very pronounced; however, considering these variable factors, I do not think that at present too much importance should be attached to the differences in the relative frequency of compensatory hypertrophy found in the series of control and of animals administered potassium iodide.

More recently Dr. J. Rabinovitch in our laboratory has determined the effect of undernourishment on the thyroid gland of normal guinea pigs. He found that when the loss of weight is great, there is a marked diminution in the number of mitoses, the acini becoming relatively small, the epithelium low and the colloid hard, thus indicating an inactive condition of the thyroid gland. It is important to be aware of these effects, otherwise one might attach to a specific factor, effects which are due to the non-specific action of loss of weight, the result of undernourishment.

In contrast with potassium iodide, when thyroid or anterior pituitary substance (Armour) is fed to guinea pigs, compensatory hypertrophy is prevented. These substances may even change the structure of the thyroid to that of an inactive gland, with relatively low epithelium, very solid colloid and relatively small acini. Mitoses are not readily seen in such an organ. Thyroxin acts like desiccated thyroid and both these preparations are somewhat more active than anterior pituitary substance. But in principle they all act in about the same manner, inhibiting the activity of the thyroid gland. So far we have used only the anterior pituitary substance of Armour and Company. There is reason for assuming that the active constituent in this case is not identical with other hormones in the anterior pituitary substance which accelerate growth and the maturation of Graafian follicles.

Quite recently, Dr. S. H. Gray, in extending these investigations, has found that the regenerative processes, which take place in the acini near a cut surface, are likewise delayed or prevented in guinea pigs receiving thyroid or anterior pituitary substance in contradistinction to control and animals given potassium iodide.

After having thus established the effect of these substances on the thyroid gland during compensatory hypertrophy, we proceeded to make corresponding investigations with the normal gland. Dr. Gray and I found that administration of potassium iodide to normal guinea pigs produces, within a period of about three weeks, an intense mitotic cell proliferation in the acinus cells. Associated with this process is a slight softening of the colloid and also a slight increase in the size of the acinus cells and probably also of the acini. At still later periods the colloid takes up more water and consequently more swelling takes place; there is, in addition, the possibility that added material is secreted into the acini. Thus the pressure in the acini increases, in late periods, and, under these unfavorable conditions, the mitoses cease.

It was an interesting problem to determine whether a quantitative relation exists between the amount of potassium iodide given and the increase in mitotic activity. Dr. Rabinovitch, who investigated this point, found that such is the case. Doses varying between 0.01 and 0.1 gm. were tested, and he noted that the larger the quantity of iodine given, the greater was the effect. While the normal number of mitoses in the thyroid gland of the guinea pig is approximately 180, after 15 to 20 daily feedings with 0.1 gm. potassium iodide the number of mitoses may increase to 6000. In general, the number of phagocytes is also much increased after potassium iodide administration.

Dr. Gray and Dr. Rabinovitch furthermore determined approximately the smallest dose of this substance which still is active. Potassium iodide 0.0001 gm. per day was found ineffective; but if 0.001 gm. was given daily there was a slight rise in the number of mitoses in some of the guinea pigs, although in the majority the number remained unchanged; in addition there was, in some animals, an increase in the size of the epithelial cells and in the softening of the colloid. It is of special interest that these structural changes can be found in glands in which the number of mitoses is not yet increased. To some extent mitotic proliferation and the other changes indicating gland activity, brought about by administration of potassium iodide, are therefore independent of each other, although usually a parallelism exists between these two sets of factors.

Preliminary experiments of Dr. Rabinovitch seem to indicate that within three days after cessation of potassium iodide administration the increased mitotic activity in the acinus cells has returned to normal.

Having thus established the difference between potassium iodide on the one hand, and thyroid and anterior pituitary substances on the other hand, it was of interest to determine whether it is possible to counteract the action of one by the

administration of the other. The first experiments of this kind were carried out in our laboratory by Dr. H. A. McCordock. He found that by feeding a guinea pig daily with a 5 grain tablet of anterior pituitary (Armour), it was possible approximately to neutralize the effect of daily doses of 0.05 gm. potassium iodide. The number of mitoses under these conditions remained about the same as in normal animals, although there was still a slight stimulating effect due to the potassium iodide, as indicated by a slight increase in the average size of the epithelial cells, in the softening of the colloid, in a number of acini, and in the phagocytic activity. We are therefore justified in assuming an antagonism between potassium iodide on the one hand, and anterior pituitary and thyroid substances on the other hand.

Similarly, Gray and Rabinovitch found more recently that addition of thyroid substance prevents the increase in mitotic proliferation caused by potassium iodide. With this combination likewise the epithelium was still higher, the colloid softer and the mitoses somewhat more numerous than when thyroid alone was fed, thus indicating that potassium iodide had not yet entirely lost its influence. Other combinations, also, were tested, such as successive administrations of potassium iodide and thyroid substance or of thyroid substance followed by potassium iodide; in the former instance, the effect of thyroid predominated; in the latter case, that of potassium iodide.

In view of such definite changes produced in the thyroid gland of the guinea pig by potassium iodide, one would have expected that administration of this substance would produce marked alterations in the basal metabolism. However, this is apparently not the case, as Dr. Cordonnier has found in recent experiments. We must therefore assume that the changes produced in the gland by iodine, which indicate a stimulation, are counteracted by another mechanism. It is possible

that potassium iodide causes a retention of the thyroid hormone within the gland. It is also conceivable that the stimulation of the thyroid gland by potassium iodide does not lead to an overproduction of thyroxin or that peripheral effects counteract its actions on the thyroid gland. Here we have before us a problem which needs further investigation.

In this connection it may be stated that several previous authors and more recently, Cole, Gray and Womack have observed that a general infection in the human organism leads to solution of the colloid and desquamation of epithelium, and similar findings were made by Cole and Womack in the thyroid of the dog. In the guinea pig, infections which Gray and Rabinovitch produced experimentally did not have such effects. The effects observed were mainly those caused by concomitant undernourishment, namely a reduction in the size of the acini and in the height of the acinus cells, and a hardening of the colloid.

Concerning the method used by us, we have introduced quantitative measurements which allow accurate determinations of the effects of various substances, either alone or in combination with each other, on the thyroid gland. As far as our results are concerned, they alter the conception which has prevailed so far as to the effect of iodine under experimental conditions; they furthermore show thyroid substance and potassium iodide do not act in an identical manner on the thyroid gland, but that on the contrary, thyroid and also anterior pituitary substance (Armour) counteract the effect of iodine. Whether the marked increase in mitotic proliferation and the increase in the size of the epithelium and the slight softening of the colloid caused by potassium iodide are coordinated effects, or whether the increase in mitoses is the result of preceding changes in the colloid remains still to be determined.

So far these conclusions apply only to the guinea pig. However great, apparently, as the discrepancies are between the effects

of potassium iodide on the thyroid gland in man and dog, on the one hand, and in the guinea pig, on the other hand, it is possible that in both cases this substance stimulates the production of colloid and the activity of the gland, and at the same time causes, at least temporarily, a retention of the secretions in the lumen of the acini. Thus it would be possible to explain the lack of increase in basal metabolism following the application of iodine and the temporary improvement following its use in Graves' disease. It is very probable that the reduction in the size of the acinus cells observed after administration of potassium iodide in the dog, and at later periods also in the guinea pig, does not indicate a primary depressing effect of this substance on the thyroid gland, but that it represents merely a secondary pressure effect caused by the increased content of the acini. The principal difference between the action of potassium iodide on the thyroid in man or dog and in the guinea pig would thus consist in the hardness of the colloid produced in the former and in its relative

softness in the latter species. In such a tentative manner we will perhaps be able to some extent to harmonize what otherwise might appear as great discrepancies in the action of this substance in different types of organisms. We may furthermore assume that man is adapted to a certain level of iodine intake. If the intake remains below this level, the thyroid gland may, under certain conditions, respond with growth processes, leading to the development of simple goiter. A deficit as well as an excess of iodine might thus act as a stimulus in the thyroid gland, a relative equilibrium being attained at an intermediate point at which enough iodine is procured to take care of the physiological needs of the organism. In order to give further insight into the structural and metabolic changes in the thyroid gland, under normal and pathological conditions, we have to find further equations; especially will it be necessary to study, by means of the methods used in the guinea pig, the thyroid glands of certain other species of animals.



CAUSES & EFFECTS OF ENDEMIC GOITER*

ROBERT OLESEN, M.D.

CINCINNATI

BECAUSE of the inconclusiveness of many of the observations relating to the causes and effects of simple goiter the United States Public Health Service has conducted several studies designed to yield information on these points. Three of these investigations were directed to the determination of whether the thyroid gland is adversely affected by communicable diseases, by routine physical exercise or by decayed teeth and diseased tonsils. Another series of three studies had for its purpose the determination of the effects of endemic goiter upon intelligence, physical growth and school attendance.

With the exception of one study conducted in Connecticut, concerning the relationship between simple goiter and potential foci of infection, all of the investigations were undertaken among the school children of Cincinnati. The general conditions, limitations and findings of each study will be considered separately.

1. THE THYROID GLAND AND COMMUNICABLE DISEASES¹

Infections and intoxications have long been believed to constitute important etiological factors in goiter production. McCarrison has been foremost in expounding the theory that goiter is a water-borne disease. His arguments in favor of the infectious origin of simple goiter may be summed up as follows:

a. Goiter incidence decreases as the water supply increases in purity.

b. Goiter has been produced by the ingestion of the residue from a Berkefeld filter through which goitrogenous water has passed.

c. Intestinal antiseptics cause the disappearance of endemic goiter.

This infectious origin of simple goiter

is supported by a number of authorities, including Crotti, who suggests that an organism of the genus *Trypanosoma* is involved. However, if the supposed infection in goiter is active in character it is, as Adami has pointed out, of a peculiar type. Such an infection differs from others in appearing at a particular age and remaining only so long as the individual remains in the goitrous region. When the person moves from this district before permanent changes occur in the thyroid function, the goiter tends to become reduced in size and may even disappear. Most American investigators consider simple goiter a deficiency disease, the malady being due to a lack rather than the presence of some substance or element in the food and drinking water.

Among the etiological factors suspected of producing simple goiter, infectious diseases have frequently been mentioned. In order to determine the existence of such a relationship a study, directed along two general and complementary lines, was undertaken in Cincinnati. The first phase of the study consisted of thyroid examinations of 589 boys and 636 girls before and after certain communicable diseases. After such illnesses no changes in thyroid size were detected among 51.7 per cent of the boys and 45.6 per cent of the girls. Thyroid size was decreased in 13.8 per cent of the boys and 11 per cent of the girls. Increased size was noted among 34.5 per cent of the boys and 43.4 per cent of the girls.

In connection with the portion of the investigation just discussed a control group, comprising 1842 boys and 1783 girls who did not have communicable diseases in the interval between two separate thyroid examinations, was included. In this group no change in thyroid size had

* Read at the Annual Meeting of the American Association for the Study of Goiter, Dayton, March 25-27, 1929.

taken place among 48.4 per cent of the boys and 43.6 per cent of the girls. Thyroid size had decreased in 12.7 per cent of the boys and 18.6 per cent of the girls, while an increase was recorded among 38.8 per cent of the boys and 35.7 per cent of the girls.

The second phase of the study dealt with the relationship between the thyroids and past communicable diseases of 7977 boys and 8441 girls. In this group there was little difference in percentage incidence among the thyroid-normal and thyroid-enlarged children who had the same maladies.

There is some evidence to show that one of the immediate effects of communicable disease among girls of elementary school age is a simple enlargement of the thyroid gland. However, this thyroid enlargement appears to be temporary in character. A comparatively short time, the length of which is as yet undetermined, after a child recovers from a communicable disease, he is no more prone to changes in thyroid size than a child who has not had a communicable disease. In so far as elementary school children are concerned, there appears to be no ground for assuming that the ordinary communicable diseases are responsible for simple goiter.

2. PHYSICAL EXERCISE AS A POSSIBLE CAUSE OF ENDEMIC GOITER

In investigating the effect of physical exercise upon the thyroid gland two separate studies were made. The first of these dealt with the effect of the physical education program in the Cincinnati High Schools upon the thyroid glands of 2996 boys and 3111 girls. In the second investigation an attempt was made to determine the effect of a season of high-school football upon the thyroids of 123 players.

Thyroid enlargements of all degrees were present to the extent of 25.5 per cent among the boys and 47.3 per cent among the girls examined in four high schools. Among the boys the total percentage of thyroid enlargement was least in the

group most active in physical education and greatest in the least active group. With certain minor exceptions these generalizations are supported by a study of the percentages of thyroid enlargements occurring at each age period between thirteen and eighteen years.

Girls who participated in physical exercise to a moderate extent had fewer thyroid enlargements than those in either the most or least active groups. A greater amount of thyroid enlargement was present among those indulging in physical exercise to a slight extent than among those whose participation was marked. These variations, while neither uniform nor marked, are further corroborated by a study of the percentages of thyroid enlargements at each age period.

Thyroid examinations of 123 football players attending high school in Cincinnati, Ohio, failed to reveal significant changes in the gland as the result of a single season of training and extra activity. Apparently the thyroid glands of football players in this city are not influenced to an appreciable extent by strenuous physical exercise. Moreover, there appears to be very little of the condition known as simple goiter among the boys who play football in Cincinnati. It may be that the absence of thyroid involvement enables certain individuals to participate more readily and efficiently in this sport than when simple goiter is present.

The attempt to learn whether physical exercise is an underlying or secondary factor in the causation of endemic goiter is typical of the difficulty encountered in research of this type. While it may be suspected or even believed that exertion by an adolescent boy or girl may be the cause of simple thyroid enlargement it is quite another matter to submit convincing evidence of this relationship.

Until more accurate information becomes available it appears unjustifiable to assert that the program of physical education, as customarily conducted in high school, is the exciting cause of simple

goiter. In all probability the enlargement of the thyroid may be due, not to one, but to a combination of causes as yet imperfectly understood. By continuing to investigate and narrow down the possible exciting factors it should be possible to secure more reliable information on the subject.

3. FOCI OF INFECTION AS A CAUSE OF ENDEMIC GOITER²

The majority of the contributions to the literature dealing with foci of infection as a cause of endemic goiter are positive in character. Some of these statements are based upon experience while others are purely presumptive in character. At the present time the question would appear to be unsettled, contradictory data being available on both sides. In view of this uncertainty a study was made by the Public Health Service for the purpose of determining, if possible, the relationship between endemic goiter and such potential foci of infection as exist in decayed teeth and diseased tonsils.

The Cincinnati Study. This investigation was divided into two parts, the first taking place in the City of Cincinnati while the other was conducted in the State of Connecticut. In Cincinnati the teeth, tonsils and thyroids of 1341 white boys and 1576 white girls attending eight schools were examined. Records were kept of slight and marked thyroid enlargements as well as slight and marked decay of the teeth. In addition there was recorded the number of apparently normal tonsils, the absence of tonsils through operation, and hypertrophy and cryptic degeneration of the tonsils. In the group studied slight and marked dental decay was no more characteristically associated with thyroid enlargement than with normal thyroid status. Furthermore the degree of thyroid enlargement appeared not to be dependent upon the amount of dental caries.

In the Cincinnati study, enlargement of the tonsils was found more frequently among boys and girls without thyroid

enlargement. No consistent evidence of correlation between cryptic tonsils and thyroid status could be detected. Based upon the data gathered during this investigation it was concluded that there was no definite relationship between thyroid condition and potential foci of infection presumably located in decayed teeth, or in enlarged or cryptic tonsils.

The Connecticut Study. During a statewide thyroid survey in Connecticut an opportunity was presented for the examination of the teeth and tonsils of 5797 boys and 6608 girls in twenty-eight localities. It was the purpose of this collateral investigation to ascertain whether there was a relationship between thyroid size and potential foci of infection presumably present in decayed teeth or diseased tonsils. On the basis of the material gathered it appeared that both slightly and markedly decayed teeth, as well as hypertrophied and cryptic tonsils were more frequently associated with thyroid enlargement than with normal thyroid conditions. These conclusions, it may be noted, differ from the negative findings resulting from the Cincinnati investigation. In explanation of this discrepancy it is suggested that thyroid enlargements in Cincinnati are probably of the endemic type, being due largely to an absolute deficiency of iodine. Consequently the possible etiological rôles of decayed teeth and diseased tonsils may be overshadowed to such an extent as not to be readily discernible. On the other hand the thyroid enlargements occurring in Connecticut are undoubtedly of the sporadic type, being produced by causes other than a deficiency of iodine. With the deficiency factor largely lacking the foci of infection influence comes more prominently to the fore, the extent of its detrimental influence being more apparent.

In view of the suggested deleterious influence of decayed teeth and diseased tonsils upon the thyroid it is desirable that oral hygiene be stressed, particularly among school children. The maintenance

of healthy teeth, through appropriate nutritional guidance and practice, as well as by competent dental prophylaxis, and treatment, is obviously indicated. Appropriate treatment for enlarged and diseased tonsils is also advisable. However, caution should be exercised in the presence of an acute inflammatory condition of the tonsils lest a general flare-up be precipitated. Because individuals with decayed teeth and diseased tonsils are thyroid-normal is no reason for withholding corrective and remedial efforts. Quite on the contrary appropriate measures are desirable both for thyroid-normal and thyroid-enlarged individuals.

4. ENDEMIC GOITER AND INTELLIGENCE³

Owing to oft-repeated, though poorly supported statements that children with simple goiter have a lower grade of intelligence than thyroid-normal individuals, a study was undertaken for the purpose of gaining information on this point.

Of the 3796 children included in this investigation some degree of thyroid enlargement was found among 25.2 per cent of the boys and 39.6 per cent of the girls. Two indices were utilized in determining the intelligence of the children: First, the information afforded by school retardation or advancement, as indicated by age; second, the records of a standard group test devised to study intelligence.

Analysis of the chronological age data, indicative of school retardation or advancement, failed to reveal significant variations between thyroid-normal and thyroid-enlarged children. A comparison of the percentile ranks of the thyroid-normal and thyroid-enlarged individuals likewise failed to show differences of sufficient magnitude to warrant the conclusion that the thyroid-normal have a keener mentality than the thyroid-enlarged.

Children with marked thyroid enlargements were apparently slightly less intelligent on the average than those with normal or slightly enlarged thyroids. However, the number of children with marked thyroid

involvement was relatively small, suggesting the desirability of further observations on children with marked thyroid enlargements before drawing conclusions concerning the influence of thyroid size upon intelligence.

5. ENDEMIC GOITER AND PHYSICAL DEVELOPMENT.⁴

For the purpose of determining the effect of endemic goiter upon physical growth 1341 white boys and 1576 white girls attending eight elementary schools in Cincinnati were examined. Some degree of thyroid enlargement was present among 38.4 per cent of the boys and among 58.8 per cent of the girls. Estimates of nutrition and posture, as well as 10 uniform physical measurements, were made of each child.

The data secured suggested that thyroid enlargement was slightly less frequent among native-born girls. The available information on this point among the boys was insufficient for the drawing of satisfactory conclusions. According to the estimates of the examiners, better nutrition and posture was slightly more frequent among thyroid-normal boys and girls.

Considerably greater percentages of thyroid-normal children were more than 10 per cent overweight than were thyroid-enlarged individuals. Conversely, underweight was more frequent among thyroid-enlarged children. There was little significant difference between the average standing heights of thyroid-normal and thyroid-enlarged boys. The average standing heights of thyroid-enlarged girls were greater than those of thyroid-normal girls. Average sitting height was consistently greater among boys and girls with thyroid enlargement.

Average chest circumferences and both transverse and anteroposterior chest measurements were slightly greater among children having normal thyroid glands. This study apparently showed, despite obvious limitations due to the inclusion of a relatively small group of children in a district of moderate goiter incidence, that

children with normal thyroid glands have a definite though not marked superiority in certain physical measurements. The findings are suggestive and indicate the need for maintaining a normal thyroid gland lest a retarding influence be exerted upon physical growth.

6. ENDEMIC GOITER AND SCHOOL ABSENTEEISM³

This investigation was undertaken for the purpose of learning the character and extent of absence from school among thyroid-normal and thyroid-enlarged children. Records were kept for one year of absences due to avoidable and unavoidable causes among 479 white and 83 colored boys and also 478 white girls and 107 colored girls in the sixth grade of eleven schools. In addition 23 white and 18 colored boys and 35 white and 25 colored girls attending open-air classes were included.

Thyroid enlargements of some degree were present among 55.5 per cent of the white girls and 65.4 per cent of the colored girls examined. Forty-two and nine-tenths per cent of the white boys and 50.6 per cent of the colored boys had thyroid enlargement. Lower percentages of thyroid enlargement were found among the children attending the open-air classes, who were younger.

Average absences from school were much greater among the colored than among the white children. Among the white children the average absences were slightly greater among the girls. The opposite condition held among the colored children. A comparison of thyroid-normal and thyroid-enlarged children showed, in the particular group under consideration, a slightly greater average absence in the former group. Common colds caused a slightly greater loss of time from school among the girls.

The average time loss of pupils in open-air classes exceeded the average absence of those attending the regular classes. Absenteeism among the thyroid-normal and thyroid-enlarged children in the open-air classes was irregular in character and without special significance, probably because of the small numbers, the lower ages and relatively small amount of thyroid involvement.

In this investigation it was demonstrated that the average school attendance, at least in this selected group, was slightly, though not significantly, better among the individuals with some degree of thyroid enlargement of the thyroid gland. It follows, therefore, that the various mental and physical ailments from which children with endemic goiter are alleged to suffer were not measurably reflected in the school attendance of the particular group studied.

CONCLUSIONS

It is difficult to fix upon any single factor the responsibility for causing endemic thyroid disease. Likewise, a specific mental or physical state cannot be regarded as the exclusive result of goiter unless evidence of irrefutable character is available. The difficulties surrounding the efforts to study the causes and effects of endemic goiter are due to the complexity of the subject, a fact that is steadily becoming more evident.

Endemic goiter is simple in name only, for the etiological factors involved in its production are undoubtedly numerous. Thus, while a deficiency or absence of the element iodine is undeniably the immediate cause of this condition, there are also certain underlying or secondary factors such as infections, intoxications, faulty food habits and demands for extra iodine in epochs of the female life that definitely influence the thyroid hyperplasia.



GRAVES' CONSTITUTION (WARTHIN)*

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THE introduction of the term "toxic adenoma" to the terminology of diseases of the thyroid gland initiated a controversy the extent of which is reflected by the numerous conflicting reports which have appeared in medical literature during the past six years. Even among those who have adhered most closely to H. S. Plummer's conception of this entity there is no unanimity of opinion as to what constitutes either the clinical or the pathological picture of the disease. Opposed to Plummer's belief that Graves' disease and "toxic adenoma" are two distinct diseases, widely different in their clinical manifestations and in their reaction to iodine therapy, is the opinion of such an excellent observer as Allen Graham, who finds no justification for such an arbitrary consideration.

In keeping with the advances in thyroid surgery during the past decade, the pathologist has had an ever-enlarging opportunity to contribute to a solution of this problem because of the abundance of surgical material which has been brought to him for study. Most pathologists have been puzzled by their inability to reconcile the various clinical concepts of Graves' disease, toxic adenoma, toxic goiter, hyperthyroidism, thyreotoxicosis, etc. with their histopathologic findings. In those cases in which definite epithelial hypertrophy and hyperplasia exist in the thyroid gland of a patient who presents the classical symptoms of Graves' disease, no difficulty has been experienced in conforming the pathological findings to the clinical syndrome. But in that large group of cases in which the clinical picture is identical but in which microscopic studies reveal no epithelial hypertrophy or hyperplasia, many pathologists have contented themselves with the belief that morphologic

criteria are not reliable in such cases. They have been further confused by the knowledge that epithelial hypertrophy and hyperplasia may be found in the thyroid glands of individuals who presented no clinical evidence of thyroid hyperfunction or dysfunction. Histologic studies based entirely on thyroid epithelial changes have not led to any solution of the problem. Most pathologists have become convinced that attempts to justify clinical findings with thyroid epithelial changes alone very frequently end in failure.

Are there, then, any morphological criteria which give unity to all of the clinical concepts of hyperthyroidism or dysthyroidism? Warthin is the first to suggest that there is. In an address, given in St. Louis in 1924, Warthin discussed "The Constitutional Entity of Exophthalmic Goiter and So-Called Toxic Adenoma." Even though the term "Graves' constitution (Warthin)" has repeatedly appeared in the literature since that time,^{1,2} it was not until December, 1928, that the original paper was published.³

Warthin states that symptom after symptom has been added to the original triad of goiter, tachycardia and exophthalmos until it has become often impossible to make a positive clinical differentiation between cases of what certain observers regard as exophthalmic goiter and what others call by such names as toxic adenoma, toxic goiter, etc. A critical analysis of the present-day broad and variable conceptions of these conditions leads Warthin to believe that all of these conditions present a clinical picture of a well-defined type of human individual—a distinct pathological constitution. Certain European writers have recognized this fact and speak of a *hyperthyroid constitution*. In the absence of positive evidence of thyroid hypersecre-

* From the Diagnostic Laboratories of the Miami Valley Hospital, Dayton, Ohio. Read at the Annual Meeting of the American Association for the Study of Goiter, Dayton, March 25-27, 1929.

tion, Warthin prefers to term it *Graves' constitution*.

A person of this diathesis usually

Sugar tolerance is frequently diminished, with the development of hyperglycemia. Allergic phenomena are common. The



FIG. 1. Typical Graves' disease, showing diffuse epithelial hypertrophy and hyperplasia, with undulating basement membranes and scant watery colloid. Poor response to preoperative lugolization. Lymphoid hyperplasia with well-defined germ centers.



FIG. 2. Graves' disease with fair response to preoperative lugolization. Some of acini are distended with thin vacuolated colloid and show flattening of epithelial cells, while other acini contain little or no colloid and are lined by tall epithelial cells. Extensive lymphoid hyperplasia with lymphoid exhaustion of hyperplastic germ centers.

presents certain characteristic stigmata. The skeleton is usually slender and delicate. The facies is that of an alert, wide-eyed, quick-reactioned individual. Muscular tremor is quite constantly present. The skin is usually warm, moist, delicate and translucent, with a tendency to excessive pigmentation; hyperidrosis and dermatographia are common. The cervical lymph nodes and thyroid gland are usually enlarged. The pulse is rapid and full; the pulse pressure is usually high. Tachycardia is common and the rate is increased by slight physical or emotional efforts. Central nervous system instability is manifested by exaggerated emotional responses, undue haste and anxiety, frequent "nervous breakdowns," and the occasional development of a true psychosis, although in most instances the mind is highly developed and well preserved. Sympathetic nervous system imbalance is a prominent feature of the constitution, as shown by vasomotor instability, so-called "hot flashes," excessive sweating and localized edemas. The basal metabolic rate is elevated. The individual consumes an excess of food but remains slender or loses weight.

blood picture usually reveals mild secondary anemia and relative lymphocytosis. Sexual excitability is commonly associated with physical inadequacy; menstrual disturbances are common. There is diminished tolerance for adrenalin, thyroid substance and iodine. Warthin sums up the clinical picture as that of an individual with juvenile morphology and rapid functional reactions. It is at once obvious that the essential clinical characteristics of either Graves' disease or so-called toxic adenoma appear in the picture just described.

It is undoubtedly true that many individuals who present but one or two of the multiple physical or functional stigmata of Graves' constitution are being subjected to thyroidectomy without justification. In such cases the pathologist, all too frequently not provided with any information as to the clinical findings, searches in vain for morphological substantiation of the clinical diagnosis.

Further proof of the underlying constitutional unity of Graves' disease and so-called toxic adenoma is found in the constant presence of hyperplastic lymph follicles

in the thyroid tissue in cases of Graves' disease and in those cases of so-called toxic adenoma which present abundant clinical evidence of hyperthyroidism. The constant presence of a hyperplastic thymus in the cases of Graves' disease or so-called toxic adenoma which come to autopsy is now well recognized by pathologists with extensive necropsy experience. In addition to this there is invariably found lymphoid hyperplasia of the spleen, lymph nodes, Peyer's patches and solitary lymph follicles of the intestine, usually showing large pale germ centers with marked lymphoid exhaustion, together with hypoplasia, chiefly medullary, of the adrenals, and cardiovascular hypoplasia. In other words, the hyperplastic lymph follicles found so constantly in the thyroid glands of patient with Graves' disease and so-called toxic adenoma are but one manifestation of a general constitutional anomaly—the thymicolymphatic constitution.

A thorough study of the tissue from 665 thyroidectomies performed at the Miami Valley Hospital during the past two years results in the following tabulation:

Simple colloid goiter without epithelial hypertrophy and hyperplasia or lymphoid hyperplasia	14
Nodular (non-adenomatous) colloid goiter without epithelial hypertrophy and hyperplasia or lymphoid hyperplasia	12
Adenomatous colloid goiter without epithelial hypertrophy and hyperplasia or lymphoid hyperplasia	252
Diffuse epithelial hypertrophy and hyperplasia and lymphoid hyperplasia (Graves' or Basedow type)	265
Nodular (non-adenomatous) colloid goiter with focal lymphoid hyperplasia	1
Adenomatous colloid goiter with focal epithelial hypertrophy and hyperplasia and lymphoid hyperplasia (mixed type)	26
Adenomatous colloid goiter with lymphoid hyperplasia but no epithelial hypertrophy or hyperplasia	95
Total number of cases	665

From this table it will be observed that the tissue from 265 resections showed diffuse epithelial hypertrophy and hyperplasia with lymphoid hyperplasia of the Graves' or Basedow (exophthalmic) type. In every instance the tissue received was

accompanied by a clinical diagnosis of Graves' disease, exophthalmic goiter or hyperthyroidism. In the 26 adenomatous colloid goiters with both lymphoid hyperplasia and epithelial hypertrophy and hyperplasia, and in the 95 adenomatous colloid goiters with lymphoid hyperplasia but with no epithelial hypertrophy or hyperplasia, diagnoses of Graves' disease, exophthalmic goiter, hyperthyroidism, toxic adenoma or toxic goiter were made. It becomes at once apparent that in all of these cases of Graves' disease or so-called toxic adenoma there is one constant histological finding—the lymphoid hyperplasia.

Of 265 autopsies performed by the writer during the past two years, 47 individuals have shown gross and microscopic evidence of status thymicolymphaticus, an incidence of 18 per cent. Among these were 5 instances of postoperative death following thyroidectomy—4 due to pneumonia and 1 the result of an acute thyroid crisis. In 3 of the 5 cases of postoperative death the clinical and pathological diagnosis was Graves' disease; in the other 2 the clinical diagnosis was toxic adenoma. The thyroid tissue in the latter 2 cases showed no epithelial hyperplasia, but extensive lymphoid hyperplasia. All 5 showed all of the associated pathologic anatomical changes of status thymicolymphaticus. The thyroid tissue of the remaining 42 thymicolymphatics showed hyperplastic lymphoid tissue in every case but 2. Such findings indicate the wide prevalence of this constitutional anomaly.

The clinical symptomatology in cases of adenomatous colloid goiter with epithelial hypertrophy and hyperplasia and lymphoid hyperplasia and those in which there was no epithelial hyperplasia, but in which there was lymphoid hyperplasia, showed no essential difference. In 16 cases in which a diagnosis of Graves' disease had been made, no epithelial hyperplasia was found, but small adenomas and extensive lymphoid hyperplasia were present. Furthermore, there was no

essential difference in the basal metabolism determinations in cases showing extensive epithelial and lymphoid hyperplasia and

glands of individuals suffering from either Graves' disease or so-called "toxic adenoma;" that these individuals belong

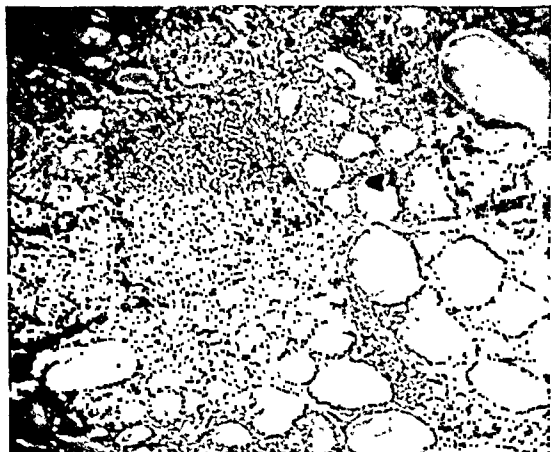


FIG. 3. Graves' disease, with good response to pre-operative lugolization. Most of acini contain a considerable quantity of thin watery vacuolated colloid, while a few of small acini show no colloid and are still lined by columnar cells. Lymphoid hyperplasia with well-defined germ centers.

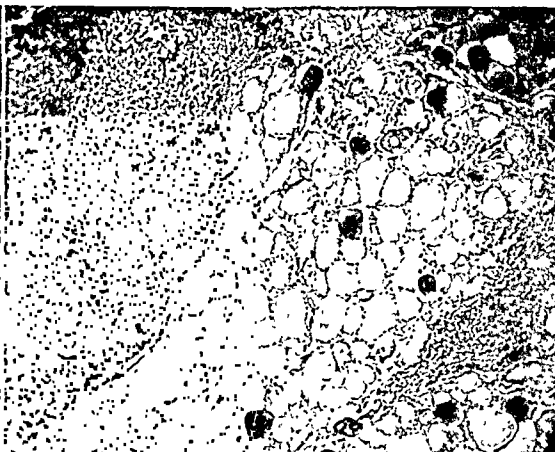


FIG. 4. Section from thyroid tissue proper in an adenomatous colloid goiter, showing no epithelial hypertrophy or hyperplasia but showing extensive lymphoid hyperplasia with well-defined germ center showing lymphoid exhaustion. Clinical diagnosis: toxic adenoma.

in those showing only lymphoid hyperplasia; in many of the latter the basal metabolic rate was plus 100.

to the thymicolymphatic diathesis; that these individuals are consequently predestined from birth to hyperthyroidism

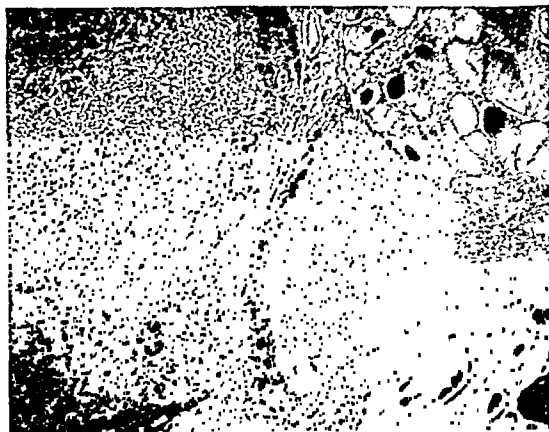


FIG. 5. Extensive lymphoid hyperplasia with lymphoid exhaustion of germ centers in adenomatous colloid goiter, showing no epithelial hypertrophy or hyperplasia. Clinical diagnosis: toxic adenoma.

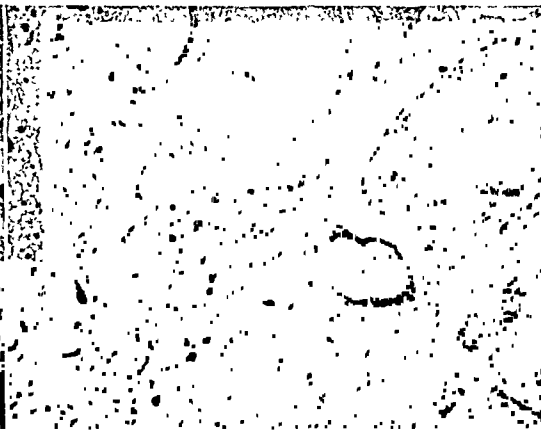


FIG. 6. Low-power photomicrograph showing extensive lymphoid hyperplasia, with no epithelial hypertrophy or hyperplasia in thyroid tissue proper of an adenomatous colloid goiter. Five lymphoid follicles with large germ centers showing the lymphoid exhaustion characteristic of thymicolymphatic constitution. Clinical diagnosis: toxic adenoma.

These findings lead to the more or less inevitable conclusions that all cases of Graves' disease and so-called toxic adenoma possess an identical underlying constitutional abnormality; that this is manifested by the constant presence of hyperplastic lymphoid tissue in the thyroid

if the necessary stimuli (physical or emotional stress, infection, etc.) are supplied; that the potentiality to the development of clinical evidence of Graves' disease or hyperthyroidism may remain

dormant for a long period or throughout life if the precipitating stimuli are absent; that the disturbance in the morphology and function of the thyroid gland is but one manifestation of an abnormality involving many other structures; that all individuals with Graves' disease or so-called "toxic adenoma" belong to the thymicolymphatic constitution, to which group Warthin has

quite aptly applied the descriptive term "Graves' constitution."

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THE HEART IN GOITER*

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HYPERTHYROIDISM, regardless of the type of goiter by which it is produced, has probably a more damaging effect on the cardiovascular system than on any of the other systems. The heart damage is progressive and the cardiac symptoms and signs become increasingly evident until they finally completely dominate the clinical picture. This is especially true in the long-standing exophthalmic goiters and the colloids which suddenly become toxic after the fourth or fifth decade of life.

The cardiac condition in goiter patients determines in a large measure whether surgical treatment should be undertaken at all, the time at which it should be done and the extent of the surgery that should be undertaken at one time. It gives some insight into the postoperative course and in some degree the extent of the cardiac recovery that might be expected after removing from the heart the burden imposed upon it by the thyroid toxemia.

A review of the literature on the cardiovascular pathology produced by thyroid disease gives us but very little information. Fatty infiltration of the heart-muscle cells has been reported as has small areas of fibrosis with perivascular round-cell infiltration scattered through the heart muscle. A few cases of extensive myocardial necrosis not accompanied by inflammatory reaction or disease of the coronary arteries have been reported. None of these findings, however, has proved constant in patients dying of thyroid disease and they are found in a number of other conditions especially as a part of the degenerative changes of old age, arteriosclerosis and chronic renal disease.

Even though we have not proved a definite cardiac pathology produced by hyperthyroidism, we do know that thyroid

disease early produces cardiac symptoms and if it exists over a sufficient period of time will so interfere with cardiac function as to produce heart failure and death.

In a review of 200 consecutive case histories of goiter patients operated on in our hospital, the following symptoms were the ones most frequently mentioned in the entrance complaint: Rapid, irregular or pounding heart, nervousness, dyspnea on exertion and fatigue or weakness. Of these 184 (92 per cent) mentioned nervousness, 160 (80 per cent) dyspnea on exertion, 154 (77 per cent) symptoms directly referable to the heart, and 140 (70 per cent) easy fatigue or weakness. Of the 154 mentioning the heart, 32 were found to have arrhythmias. Of these, 24 had auricular fibrillation, 6 had ventricular extrasystoles, and 2 attacks of paroxysmal tachycardia. The ages of these patients ranged from fifteen to seventy years. Of the 32 patients with cardiac arrhythmias the youngest was twenty-four, the vast majority occurring after the age of fifty.

The patients in this series with exophthalmic goiter all mentioned cardiac symptoms. Tachycardia was present, often for some time before the patients knew they had a goiter or any eye changes had been observed. It was noticed that in those patients under thirty years of age in whom the exophthalmic goiter developed rapidly on a simple preexisting colloid, their hearts bore the thyroid toxemia exceedingly well. They often maintained a pulse of 140 to 160 per minute for months without developing any cardiac arrhythmia or signs of congestive heart failure. In all of the young individuals who developed arrhythmias evidence of previous cardiac damage could be obtained.

In the old exophthalmic goiter patients, either those with long-standing exophthal-

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mic goiters or those in which exophthalmic goiter developed in a long-standing diffuse of nodular colloid goiter after the fourth or fifth decade of life, arrhythmias, especially auricular fibrillation, were much more frequently found. Their frequency increased in direct proportion to the ages of the patients. The auricular fibrillation appeared first in transient attacks which the patient often described as attacks of palpitation and many were aware of the cardiac irregularity. Auricular fibrillation tends to become constant and when it does the signs of congestive heart failure may appear very quickly or the heart may fibrillate for some time without failure. Auricular fibrillation may become so permanently established that regular rhythm cannot be restored after thyroidectomy even after the use of digitalis and quinidine.

The colloid goiters, both the diffuse and the nodular, may exist for as many as thirty or forty years without giving rise to any symptoms referable to the heart. They may, however, undergo a change in the character of the acinal cells with the formation of new acini in the interstitial tissue. The acinal epithelial cells tend to change from the flat pavement type to cuboidal or columnar, and toxic symptoms begin to manifest themselves. We then have the development of the toxic adenomatous goiter. These goiters becoming toxic after the fourth or fifth decade of life are the ones which produce the most marked cardiac symptoms. It is in these that auricular fibrillation and congestive heart failure so frequently occur.

In this class of patients the cardiac symptoms frequently entirely overshadow the other symptoms of thyroid toxemia. They often have only small goiters which have been present for many years and frequently they are not aware of the existence of thyroid enlargement. Many times there is more gland present than appears on ordinary examination or superficial inspection. The thyroid is often entirely overlooked as a cause for their condition. They present all the evidence

of extreme cardiac failure and have often been given digitalis for months without improvement.

The diagnosis sometimes presents a little difficulty. If there is a history of recent growth in a long-standing goiter or if eye signs are present the diagnosis is readily made, but the majority of them do not show eye signs. Marked loss of weight is the rule and if there is a history of marked weight fluctuation without apparent cause, it should make one suspicious of periods of thyroid toxemia followed by remissions. The basal metabolism is relatively lower than in the primary exophthalmic goiter. The readings run from +20 to +40 whereas in the exophthalmic, they run from +60 to +90 or even more. The flushed and perspiring skin of the young exophthalmic goiter patient is often absent and there is often marked apathy in contradistinction to the restlessness and nervousness seen in the exophthalmic goiter patients. Finally if there is a history of transitory attacks of cardiac irregularity with or without the symptoms of congestive heart failure which have been relieved without treatment, it would strongly suggest a toxic thyroid crisis followed by remission.

The fetal adenoma probably causes the least amount of cardiac damage. They are found singly or sometimes more than one in an otherwise normal gland, as small nodules in the simple goiters of youth or in combination with any of the other types of goiter. They may exist for years, gradually getting larger, without producing any symptoms or they may at any time become toxic causing nervousness and mild tachycardia. Frequently the large fetal adenomas in patients at about the age of fifty or over produce rather marked cardiac symptoms. Arrhythmias and congestive failure often develop. When these are removed and examined microscopically the changes seen are often degenerative rather than proliferative.

There are several types of patients with cardiac symptoms and goiter in which the

goiter is merely incidental and has nothing to do with the cardiac symptoms. Thyroidectomy is worse than useless in these patients.

One of these is the young frail-appearing female, with a small, firm, uniform goiter, who usually complains of palpitation and dyspnea on exertion, ease of fatigue, nervousness, and choking, the latter especially if someone has told her that she has a goiter. They usually have dysmenorrhea and often give a bizarre symptomatology of a neurasthenic nature. They often have had their appendix removed, their cystic ovaries punctured or partially removed and their uterus suspended without results. Their cardiac rate is often found to be 100 or over. The rate increase is not constant, however, becoming normal on rest and sedative medication. It is also normal while asleep. These latter findings together with the multiplicity and nature of their symptoms, the absence of marked weight loss and a normal basal metabolism make their differentiation from toxic goiter rather simple. These are cases of so-called neurocirculatory asthenia.

Another type comprises the patients over fifty years of age with a systolic blood pressure of 180 to 200 and a small, firm, uniform or nodular goiter. They complain of rapid heart and dyspnea on exertion. The heart is often enlarged and there may be signs of congestive heart failure. Auricular fibrillation or other types of arrhythmia may be present. These are cases of essential hypertension, or the examination of their urine and the estimation of their blood creatinin and non-protein nitrogen may show definite evidence of renal disease and insufficiency.

The differentiation of these from the toxic adenomatous goiter is not always easily made. Their basal metabolism is practically normal, both systolic and diastolic blood pressures are equally elevated and their heart rate is more easily reduced on rest and proper elimination.

Finally there is the patient past middle age with a nodular non-toxic goiter with serious heart disease and definite evidence of failure. The basal metabolism is normal and the failure cannot be attributed to the goiter. They must be classified as chronic myocarditis often of unknown etiology.

None of these three types is benefitted by thyroidectomy and surgery is definitely contraindicated.

SUMMARY

1. Thyroid toxemia if acting sufficiently long always produces cardiac symptoms and eventually serious heart damage.
2. There is no definite cardiac pathology directly attributable to thyroid toxemia.
3. Undamaged hearts in young individuals bear thyroid toxemia exceedingly well.
4. Thyroid toxemia frequently produces cardiac arrhythmias, auricular fibrillation being the one most frequently seen.
5. Toxemia developing in long-standing colloid goiters produces a clinical picture in which the cardiac symptoms completely overshadow the other symptoms of toxemia.
6. There are three types of patients with goiter and cardiac symptoms and findings, namely, neurocirculatory asthenia, hypertension cases and those with myocarditis in which the goiter is only incidental and in whom thyroidectomy is definitely contraindicated.



SURGICAL RISK IN HEART GOITER CASES*

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THE preoperative and postoperative administration of iodine having to a large extent lessened postoperative hyperthyroidism and death from this cause has enabled the surgeon to undertake surgery in a wider range of bad heart cases due to goiter than formerly. The fact that the largest mortality occurred with these patients suggested a study of such methods as would make the operation less hazardous.

In considering the goiter bad heart as a surgical risk, it is assumed the heart has been improved to the fullest extent by preoperative treatment. The chief class of cases for discussion is the one that notwithstanding such maximum improvement has, however, the left cardiac border near the midaxillary line, an irregular cardiac impulse of about 120 with a radial pulse of 100 or more, and has associated with it more or less edema extending upward to about the hips. In these cases the cause of postoperative death frequently is added cardiac dilatation, edema of the lungs and pneumonia. An additional class of cases is those with a hypertension of 200 mm. Hg or more in which the cause of death frequently is acute dilatation of the heart or apoplexy. In our experience the latter class occurs more often in the elderly patient with toxic adenoma.

To reduce the risk to the heart, apparently the objective of the surgeon should be threefold, as follows: (1) To employ such methods as would minimize cardiac strain caused by increased blood pressure, accelerated pulse rate, psychic disturbances, physical effort, and postoperative hyperthyroidism. (2) To sustain the heart muscle by adequate oxygenation and nourishment. (3) To minimize obstructive respiration of a somewhat embarrassed lung.

The disturbing apprehensions and other

psychic manifestations which arise just prior to operation are allayed better by hypodermics of pantopon than by morphine since the former is followed by less respiratory depression and less subsequent nausea or vomiting. To be used in conjunction with pantopon, scopolamine in ampules seems preferable to hyoscine as it is more dependable in its effect.

Systematic observations have led us to infer that local anesthesia with early infiltration of the thyroid poles is the preferable operative anesthesia. To ascertain cardiac strain during the operation we made observations of blood pressure, cardiac impulse and radial pulse rate in a series of 350 thyroid operations on all types of goiter done under local, nitrous oxide and ethylene anesthesia. In this work I am greatly indebted to my anesthetist, Dr. C. E. Ragan, for taking and charting the readings which were taken on the operating table just before preparation of the patient, five minutes after established anesthesia, during the dissection, ligation and severing of the superior thyroid poles and the interval between these procedures. A study of these readings revealed that in local anesthesia there was rarely a rise in blood pressure at the reading taken at established anesthesia whereas with the use of nitrous oxide or ethylene anesthesia there was a rise of 10 to 15 mm. Hg in the majority of instances. In a striking manner the readings disclosed in 70 per cent of the cases, under either local or general anesthesia, a rise of 12 to 40 mm. Hg during the dissection, ligation and severing of the first pole, followed by a drop almost to the preceding level. Then, during a similar procedure on the second pole there was a moderate rise followed by a drop to or below the original level. With the fluctuation of blood pressure was an apparent

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parallel rise and fall of cardiac rate. This observation led us, as soon as the pole could be identified, to infiltrate it with procaine until it blanched, thereby anesthetizing the nerve supply and blocking the escape of thyroid secretions by the intracapsular pressure. When this was done it was noted that no particular rise in blood pressure or pulse rate occurred during the manipulations about the superior poles.

These observations imply that local anesthesia with early infiltration of the poles causes less cardiac strain. It therefore seems to be the anesthetic of choice. Particularly is this true in the hypertension cases when the blood-pressure rise of 10 to 40 mm. Hg might be sufficient strain in the vessels to produce apoplexy. Yet, when the preoperative narcosis has not been sufficient to allay the psychic features, a light nitrous or ethylene anesthesia with a large mixture of oxygen should be used to supplement the local one. It has been our observation that cases done under local anesthesia have less prostration, headache, wound pain and emesis and therefore are more able to partake of fluids immediately and consume food within a few hours. Should there be any cyanosis oxygen should be administered, which also very frequently lowers the pulse and makes it more full. The adequate oxygenation of the blood and early intake of food are important factors in sustaining the myocardium.

The operation should be of the type that can be easily abandoned with a minimum amount of traumatism proportionate to its extent and productive of the least hyperthyroid reaction. Therefore, the multiple stage operation is well suited to this class of cases. It should be done with the view of attempting a lobectomy, removing first the adenomatous lobe should the case be one of mixed goiter. The resection should begin with ligation of the superior pole so that in the event of abandoning the operation for the day, something has been accomplished in reducing thyroid function. Next the lateral

veins should be ligated and severed. Then the usual resection is performed, taking great care to guard against traumatism to the bed of the recurrent laryngeal nerve and undue traction on the dislocated lobe especially in the vicinity of the nerve's entrance between the cricoid and thyroid cartilages. Even a partial paralysis from stretching or subsequent edema of the nerve may have most unpleasant consequences. If the isthmus is removed a layer of thyroid tissue should be left covering the trachea to prevent the formation of tracheal mucus. Failure to observe these small technical details may lead to impaired function of the vocal cords and excessive mucus in the trachea. Both interfere with inspiration and proper oxygenation of the blood, a very necessary function in sustaining a damaged heart muscle. In addition the tendency toward vacuum formation in the lungs during obstructed inspiration tends toward a pulmonary edema. The excessive mucus not only is likely to induce pneumonia in a lung with more or less passive congestion, but the coughing necessary to expel it brings about undue cardiac strain. The drainage provisions should be larger than usual so that there may be no absorption of wound secretion to add to the increased temperature and pulse rate of the expected postoperative hyperthyroid reaction. The time of undertaking the second lobectomy is determined by the degree and length of reaction, the cardiac and respiratory conditions, and the ability to take fluids and nourishment. The indications may become satisfactory within twenty-four hours but they usually require one to two weeks, and rarely they are delayed two months. The shorter the period between the first and second operations, the less likely will one be able to secure a good local anesthesia. As a rule the patient has made a general and cardiac gain sufficiently great so that a nitrous oxide or ethylene anesthesia will not be contraindicated except in the marked hypertension cases.

ETIOLOGY & PROPHYLAXIS OF ENDEMIC GOITER*

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THE etiology of endemic goiter cannot be regarded as completely elucidated. The iodine deficiency theory has of late won numerous adherents. And although there is no doubt that a relative lack of iodine must logically produce diseases in the thyroid gland if the supply of iodine is too scanty to cover the requirements of the thyroid tissue in the production of thyriodine, yet it is equally certain that lack of iodine cannot be the only cause of endemic goiter.

The fact, however, that all the causes which increase the need of the system for iodine, such as puberty, lactation and climacteric periods favor the appearance of goiter, speaks strongly in favor of the iodine deficiency theory. In any case we can say with confidence that lack of iodine may be regarded as one of the causes of endemic goiter and most likely the primary one.

I will not at the moment go further into the various questions connected with the etiology of goiter, but will merely call attention to a series of experiments which I have conducted and which may possibly throw light on the question of etiology.

Let me first call to mind the fact that the French pharmacologist, A. Chatin, demonstrated by experiment that the frequency of goiter and the concentration of iodine in the surrounding nature were in inverse proportion to each other. As is well known, his works did not receive the attention they deserved and they had therefore all too little influence on the development of the preventive treatment for goiter.

Later investigations have proved the correctness of Chatin's assertion. Von Fellenberg, for instance, has shown that the concentration of iodine in the surrounding nature is lower in the strongly endemic

districts in Switzerland than in districts where goiter is less frequent. At the same time J. F. McClendon, with his collaborators, A. Williams and J. C. Hathaway, demonstrated that there was an inverse ratio between the iodine content in drinking water and the percentage of goiter in the drafted men in the United States army. They found also a smaller iodine content in the local foodstuffs from the goitrous districts than in the food from non-goitrous districts.

A number of important investigations by C. E. Hercus and his collaborators, published about the same time as the works just mentioned, have shown that the same ratio holds good in the case of New Zealand.

If we accept the iodine deficiency theory as correct it is obvious that there must exist a minimum iodine requirement for human beings. This iodine requirement may, of course, vary with locality and climate and it may moreover be different at various periods in the life of the individual. We know for instance, that just those factors that intensify the organisms' need for iodine, such as puberty, pregnancy, lactation and climacteric periods, also increase the tendency to goiter.

Without accepting the iodine deficiency theory as absolutely correct it is still clear that there must be a minimum iodine quantity without which the organism cannot produce sufficient quantities of the iodine containing secretion (hormone) in the thyroid gland. It is naturally of the utmost importance to determine the amount of this minimum quantity of iodine.

We might adopt the method of determining the iodine content in all the foodstuffs in goitrous and non-goitrous districts and thus arrive at the average figures for

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the quantity of iodine which the food consumed daily places at the disposal of the organism.

Most careful investigations of this kind have been made by von Fellenberg in Switzerland, J. F. McClendon at the University of Minnesota and by C. E. Hercus and K. C. Roberts in New Zealand. McClendon calculated the intake of iodine through food stuffs in a goitrous district in the United States at 19 gm. per day. In New Zealand Hercus and Roberts calculated the mean iodine intake per person per day at 20.15 gm., in a goitrous and 34.85 gm. in a non-goitrous district. Von Fellenberg determined the iodine intake per individual per day in the goitrous district of Signau at 13.0 gm. and at 31.3 gm. in the almost non-goitrous district of La Chaux-de-Fouds.

Such investigations, however, take time and still give no complete picture of the daily total iodine intake. Iodine sources from which the iodine needs of humanity are supplied include the atmosphere, fresh water, animals, marine animals, and plants, the latter receiving iodine from the soil.

We must reckon with no small quantities in drinking water and in the atmosphere. It is especially difficult to determine the quantities of iodine that are inhaled daily. Another difficulty lies in the fact that not all the iodine contained in the foodstuffs consumed is resorbable and it is, of course, only resorbable iodine that interests us in our attempt to determine the minimum iodine requirement.

Instead of investigating the quantities of iodine intake we can investigate iodine excreted. Von Fellenberg, for example, determined the iodine excretion through the urine for the inhabitants of goitrous and non-goitrous districts. He found for Effingen (1 per cent goiter) an average iodine excretion of 70 gm. per individual per twenty-four hours, for Kaisten (61.6 per cent goiter) 19 gm. per individual per twenty-four hours and for Hunzenschwil (56.2 per cent goiter) 18 gm. per individual per twenty-four hours. He found also a

considerably lower average iodine content in the surrounding nature in districts where goiter percentage was highest. It must be remembered that all the iodine excreted is not excreted through the kidneys, but even if the excretion figures we get by determining the iodine content of the urine are undoubtedly too low, still they give good material for comparison.

In collaboration with Prof. Johan Nicolaysen of Oslo, I have examined the excretion of iodine through the urine in the case of inhabitants of the Norwegian goitrous district, Sandsvaer. Sandsvaer lies west of the Oslofjord and runs inland beginning some few kilometers from the coast. Goiter is here endemic. Investigations regarding the incidence of goiter among school children have been carried out by Professor Nicolaysen.

In order to determine the daily iodine excretion in the district as a whole, from 5 to 10 healthy adults from the various school districts were selected for experiment. Men only were chosen for the investigation for, as von Fellenberg has shown the excretion of iodine is not always constant in the case of women due to the facts before mentioned that women under certain conditions show an increased need of iodine. We must also bear in mind that none of the persons examined had goiter and must take this into consideration when we compare our results with those of von Fellenberg. Our aim was not to determine the excretion of iodine for goitrous persons, but to get an average of the daily excretion of iodine in normal persons in that district.

Experiments recently carried out in collaboration with Johan Holst showed that there was a great difference in the rate of excretion of iodine in the case of goitrous persons, compared with people who are normal, and that the total iodine metabolism is also quite different.

Table 1 shows the average excretion of iodine through the urine compared with the figures for the incidence of goiter in school children in the goitrous district of Sandsvaer.

TABLE I

RELATION OF INCIDENCE OF GOITER AMONG SCHOOL CHILDREN IN SANDSVAER, NORWAY, TO EXCRETION OF IODINE

District	Goiter in Per Cent	Excreted Iodine in Gms. in Twenty-four Hours
Hostvedt	59.7	40
Vittingfoss	55	48
Ljterud	54	29
Meiera	53.6	39
Saggrenda	43	64
Komnes	39.7	48
Berg	38.6	56
Eftelt	36.6	65
Verp	36	87
Ruud	30	61
Vik i Sogn	0	173

The figures for the iodine excreted in twenty-four hours were arrived at in the following way: The iodine content of night urine was determined and from this the excretion of iodine in twenty-four hours was calculated on the assumption, which is approximately correct, that a similar amount of iodine is excreted every hour.

In comparing the figures for the excretion of iodine in adult men with the figures for goiter, we must take into consideration that the latter figures refer to school children, who naturally use a smaller quantity of foodstuffs and whose bill of fare moreover is somewhat different.

Table I shows that on the whole the amount of iodine excreted falls in proportion to the rise of the goitrous percentage. For the sake of comparison I have also included figures for the non-goitrous district, Vik i Sogn. These figures were found by von Fellenberg when he examined samples of urine that Johna Holst at my suggestion had sent to him. For comparison with the figures in Table I, I add the figures which von Fellenberg found for the excretion of iodine in Switzerland and on the Ligurian coast (Table II).

In comparing von Fellenberg's figures with those for Norway, we must take into consideration that the percentage of goiter for the three Swiss villages refers to the whole population, while the Norwegian

TABLE II

INCIDENCE OF GOITER IN THE POPULATION IN SWITZERLAND IN RELATION TO EXCRETED IODINE

District	Goiter in Per Cent	Excreted Iodine in Gms. in Twenty-four Hours
Effingen	1	70
Kaisten	61.6	17
Hunzenschwiel	56.2	18
Forte dei Marmi	0	112

figures deal only with the incidence of goiter among school children.

We come therefore to the following conclusion: Our investigations have proved that the quantity of iodine excreted through the urine in adult males in Norwegian goitrous districts, is approximately in inverse ratio to the incidence of goiter among school children in the same districts.

PROPHYLAXIS

If we are now agreed that one of the fundamental factors in the spread of endemic goiter is a deficiency in the intake of iodine, the question of prophylaxis will naturally be how to abolish this deficiency, that is to say how to increase the intake of iodine.

May I in a few words review the history of the prophylaxis of goiter? I will here confine my remarks exclusively to pure prophylaxis and will not enter into questions of therapeutics.

Prevost (Geneva) in 1849 prescribed iodine as a prophylactic for goiter. His method was tried in France and somewhat later in Austria and Italy, but as we know, it was discredited as the doses used were too strong.

Not until 1909 did the question again come into prominence, when D. Marine and C. H. Lenhart showed that the so-called carcinomas of trout were in reality enlarged thyroids and could be prevented by putting small quantities of iodine in the water. In 1917 D. Marine and O. P. Kimball, as we know, commenced the preventive goiter treatment for school

children in Akron, Ohio. These attempts and their excellent results are so well known that I shall not linger over them.

In Switzerland the preventive treatment of school children with small quantities of iodine was again resumed in 1919. I may mention names like Eggemberger, Hunziker, Klinger and Roux.

Heinrich Hunziker in 1915 published his remarkable paper: "Goiter, the system's adaptation to iodine-deficient food" after he had practiced goiter prophylaxis on his patients for several years.

In Norway the preventive treatment of goiter with small quantities of potassium iodide was introduced by J. Nicolaysen.

As is well known the so-called iodized salt has been introduced into several goitrous districts, to reach thereby not only school children, but the whole population. The iodine content in the iodized salt is variable (in Switzerland 0.038 mg. in 10 gm. salt, which is taken to be the daily requirement of each individual).

Instead of using iodized salt attempts have been made, especially in America, to add small quantities of iodine to the drinking water; for instance, by B. C. Little and J. A. Goodfellow in Rochester, New York, Ilkeston and Heanor, and by H. A. Sherman in Sault St. Marie, Michigan. This method is much more expensive than the use of iodized salt.

All over the world the preventive treatment of goiter with iodized salt has shown a reduction of the percentage of goiter in school children.

There are, however, certain difficulties in the way of a general use of iodized salt. Some people have qualms about its use and it is true that the ionized iodine in that *form* in which it appears in iodized salt may really be dangerous for goitrous persons, if it were not that the addition of the salt prevents the use of too strong doses. Ionized iodine may produce toxic symptoms (secondary toxic goiter, struma Basedowifacata). There are, however, really very few cases, if on the whole there are any, where the small quantities of iodine

contained in iodized salt have really produced exophthalmic goiter. But, as I have said, many people are hesitant about its use.

Another unfortunate characteristic of the iodized salt is that its iodine content is not always constant. Von Fellenberg, for instance, has demonstrated that the iodine content undergoes a change when the salt is kept in store.

IODINE EXCRETION IN SEA-FISH CONSUMERS

Our researches at Sandsvaer, however, have led us to another method of goiter prophylaxis. When we take the iodine excretion figures for the inhabitants of the Sandsvaer district one by one, we note the fact that the quantities of iodine excreted in twenty-four hours are, in the case of some persons, very high; in the case of others very low. Some of the figures are exceptionally high. During my investigation of the urine, J. Nicolaysen examined the food lists of the persons tested and on comparing our results we found that the persons with great iodine excretion almost without exception, had eaten sea fish during the previous week. He could also show that this district had been regularly supplied for some time with sea fish and that the consumption of sea fish was on the increase.

Table III shows what a very great quantity of iodine is excreted by consumers of fish. When we take into consideration the fact that most of the persons examined had eaten no fish at all on the day before the urine test, we see that we are not dealing with an isolated accidental increase in the quantity of iodine excreted, but of an increased iodine metabolism, caused by the use of fish food. These results show us the simplest and most natural means of goiter prophylaxis, at least as far as Norway is concerned. We have only to increase the consumption of fish in the goitrous districts and to facilitate the distribution of fish-products in the in and districts.

The advantages of such a prophylaxis are manifest. Iodine is contained in the

TABLE III

RELATION BETWEEN EXCRETED IODINE AND QUANTITY OF SEA FISHES IN THE DIET

No.	Excreted Iodine in Gms. in Twenty-four Hours	Sea Fishes in the Food	No.	Excreted Iodine in Gms. in Twenty-four Hours	Sea Fish in the Food
1	42		26	128	++
2	57		27	36	+
3	62		28	6	
4	25		29	52	+
5	10		30	101	(?)
6	41		31	108	++
7	19	+	32	35	+
8	22		33	140	++
9	28		34	50	
10	36		35	37	
11	38	++	36	69	+
12	55	++	37	23	
13	50		38	31	
14	105	+++	39	79	(?)
15	212	+(KJ)	40	56	
16	14	+	41	49	+
17	67	+++	42	73	+
18	118	++++	43	49	
19	39	+	44	86	+
20	20	+	45	43	++
21	83		46	27	
22	11		47	13	
23	214	+(KJ)	48	73	++
24	59	+			
25	101	+			

fish, as we have been able to show, in a natural organic combination and in the organism it changes, very possibly gradually, into the active ionized form. This is seen clearly from the fact that the excretion of iodine by consumers of fish was shown to be high, even when they had eaten no fish the day before while, as we have been able to show, ingested inorganic iodine leaves the body within twenty-four hours. By means of fish prophylaxis the necessary iodine is obtained in a way that is better suited to the needs of the organism.

F. de Quervain, after having pointed out the large quantities of iodine consumed with their food without any injury by the dwellers of the coast, writes as follows:

It is therefore a great question whether the form of the iodine does not play a certain part. With our food the iodine comes to us predominantly in organic combinations, while in the case of iodized salt we get it in active, ionized form. It is therefore not unreasonable to sup-

pose that this active form, although no doubt more effective as regards goiter prophylaxis, yet in spite of there being only physiological quantities to hand may cause toxic effects in a population, which on account of the presence of goiter is especially sensitive.

The two Austrian physicians, J. Wiesel and J. Kretz, are also strongly in favor of goiter prophylaxis by means of iodine in natural organic combinations. They write as follows:

The ionized, inorganic iodine is extremely effective (we have only to look at the effect of the tiniest dose of Lugol's solution, or the effectivity of iodized salt. The organic iodine is first decomposed in the body in a way that probably corresponds to the natural requirement of the organism, and only gradually changes into the active, ionized form. In agreement with this supposition we have the fact that non-ionized, organically combined iodine such as especially occurs in plants and in animal organisms, never leads to iodine intoxication, in spite of the fact that this iodine is ingested in quantitatively larger amounts than, for instance, in the iodized salt. Neither has there ever been known a case of persons moving from an iodine-deficient goitrous district to one that is non-goitrous and richer in iodine getting symptoms of iodine intoxication.

After a lecture which I delivered before the society of physicians in Vienna, J. Wagner-Jauregg expressed himself strongly in favor of the use of fish products as a prophylactic against goiter.

He said:

The use of sea-fish would be a very desirable prophylaxis against goiter, and it would be a very excellent thing if physicians would make propaganda for the use of sea-fish, especially for young people with goiter, or who are in danger of getting it, and also for pregnant women.

IODINE CONTENT OF FISH AND FISH PRODUCTS

The next point is the very great importance of a study of the iodine content of the various fishes and fish products, in order that we may be able to form an idea of what quantities of iodine are at our dis-

TABLE IV

AVERAGE IODINE CONTENT OF FLESH OF NORWEGIAN TELEOSTS (MG. PER. KG.)

	Fresh (Moist)	Water- free
Haddock (<i>Gadus aeglefinus</i>).....	7.65	34.17
Cod (<i>Gadus morrhua</i>).....	4.89	22.20
Coal fish (<i>Gadus virens</i>).....	2.65	13.37
Ling (<i>Molva molva</i>).....	4.80	23.16
Cusk, torsk (<i>Brosme brosme</i>).....	3.60	17.85
Plaice (<i>Pleuronectes platessa</i>).....	0.71	3.61
Red fish (<i>Sebastes marinus</i>).....	1.41	6.60

TABLE V

IODINE CONTENT OF NORWEGIAN STOCKFISH AND SPLIT COD (MG. PER KG. WATER-FREE SUBSTANCE)

	Fresh Fish	Dried Fish	Split Cod
Cod (<i>Gadus morrhua</i>).....	22.20	16.6	6.8
Haddock (<i>Gadus aeglefinus</i>).....	34.2	30.6	
Coal-fish (<i>Gadus Virens</i>).....	13.4	4.8	

TABLE VI

IODINE CONTENT OF NORWEGIAN COD-LIVER OIL (OLEUM JECORIS ASCELLI) (MG. PER KG.)

Cold-cleared oil.....	15.200
Row medicinal oil.....	14.200
Pale medicinal oil.....	9.900
Light brown oil.....	4.500

posal. P. Bourcet examined various kinds of sea fish and found in all large quantities of iodine. Before closing our investigations a number of results obtained by D. K. Tressler and A. W. Wells of the U. S. Bureau of Fisheries regarding the iodine content of various fish products came to our knowledge. N. D. Jarvis, R. W. Clough and E. D. Clark had examined the iodine content in Pacific Coast salmon and recommended this also as a prophylactic against

TABLE VII

IODINE CONTENT OF NORWEGIAN SARDINES IN OLIVE OIL. BRISLING (*CLUPEA SPRATTUS*) (MG. PER KG.)

Brisling		Olive Oil		Total Content of Can	
Moist	Water-free	Moist	Water-free	Moist	Water-free
0.83	1.97	0.75	0.77	0.21	0.45
1.28	2.85	0.75	0.76	1.19	2.22
1.50	3.51	1.29	1.34	1.44	2.64

TABLE VIII

IODINE CONTENT OF NORWEGIAN KIPPERED HERRINGS (*CLUPEA HARENGUS*) (MG. PER KG.)

Herring		Liquor in Can		Total Content of Can	
Moist	Water-free	Moist	Water-free	Moist	Water-free
0.38	1.08	0.50	0.77	0.33	0.93
				0.39	1.04
				0.51	1.43
0.53	1.44	1.05	1.30	0.67	1.64
1.29	3.55	0.50	2.99	1.26	3.54
1.89	4.95	0.57	1.06	1.86	4.81

goiter. Some stray determinations of iodine in fish products were also available. In collaboration with H. Haaland and S. O. Madsen I examined some species of Norwegian teleosts with the results shown in Table IV. Karl Closs and I examined Norwegian dried fish and split cod, both of which showed a somewhat lower iodine content than the fresh fish (Table V). The endemic districts, however, lie far inland and the transport of the fresh fish is difficult and expensive. We have therefore

TABLE IX
IODINE CONTENT (MG. PER KG.)

Kind of Preserved Sea Food	Meat		Liquor in Can		Total Content of Can	
	Moist	Water-Free	Moist	Water-Free	Moist	Water-Free
Herring (<i>Clupea harengus</i> in olive oil.....)	0.75		1.51		0.92	
Herring, smoked.....	0.75	1.99	0.79	0.83	0.77	1.47
Herring, smoked.....					0.31	0.60
Mackerel (<i>Scomber scombrus</i>).....	0.85	2.22	1.90	20.70	1.09	3.44
Roe of cod (<i>Gadus morrhua</i>).....	1.92	6.84	2.51	27.87	2.01	8.03
Fish-balls (<i>Gadus aeglefinus</i>).....	1.06	6.60	0.42		0.82	7.74

examined a number of samples of canned fish goods and in these also we detected the presence of great quantities of iodine. These researches were principally carried out in collaboration with Sigurd Ostad Madsen. Tables VI to IX show the result of our researches.

The quantities of iodine determined in the canned fish examined by us are so great that a reasonable quantity in the weekly bill of fare is sufficient to cover the iodine requirements of any organism even in strongly endemic districts.

I have emphasized the importance of the iodine being supplied in organic combination. It is therefore of the greatest interest to find out in what organic combination iodine exists in fish foods. I have collabo-

rated with Karl Closs in this study and we examined especially Norwegian split cod and some other fish products. We were able to demonstrate the presence of iodine in combination with albumens and also with fats or lipoids, and in other organic combinations. Some very small amount was present in inorganic form. These researches into the nature of the iodine compounds in fish products are being continued.

I will conclude by saying that in fish products we have a useful and simple means of supplementing the iodized salt prophylaxis. In the fish products examined and especially in the easily transportable canned goods, we determined a high content of iodine in organic combination.



INTERMEDIATE IODINE METABOLISM DURING THE PREOPERATIVE TREATMENT OF EXOPHTHALMIC GOITER*

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OSLO, NORWAY

THE following investigations were carried out on patients of surgical clinic A of the University of Oslo. The clinical investigations, determination of the metabolism figures and histological investigations were carried out by Johan Holst. He also operated upon the patients and took samples of blood. The chemical analyses of blood and thyroids were made by G. Lunde in collaboration with Karl Closs and O. Chr. Pedersen in the Pharmacological Institute. The method used for the determination of the blood iodine was worked out by Gulbrand Lunde on the basis of the method suggested by von Fellenberg.

The preoperative treatment with Lugol's solution in the cases here examined was as follows: The patients were given two to four times daily for ten to twenty days 10 drops of a solution of the following composition:

Sol. Potassium Iodide (10 per cent)	100 parts
Iodine	5 parts

According to the clinical reaction to this treatment we were able to divide our cases into the following three groups:

Group I. Typical reaction. In the course of the Lugol treatment, clinical improvement until the patient was free of symptoms. Falling of metabolism figure to, or almost to, the normal.

GROUP II. Slow reaction. After an initial, incomplete improvement, exacerbation in spite of (or on account of?) continued iodine treatment.

GROUP III. Doubtful, or no effect of the preoperative iodine treatment.

Of the 32 cases treated during the last

year, 24 belonged to Group I, 5 to Group II, and 3 to Group III.

The histological investigation of the primary toxic goiters (genuine exophthalmic goiters) removed from patients treated with Lugol's solution showed, apart from diverse alterations of the cells which we may overlook for the present, a *strikingly larger amount of colloid* than the untreated primary toxic goiters. The untreated primary toxic goiters showed a thin thread-formed colloid, difficult to color with eosin. In contrast to the structure of these untreated thyroids, those treated with the Lugol's solution showed a homogeneous, normal or almost normal-looking colloid easily colored with eosin. With regard to the relation of the colloid before and after the treatment of primary toxic goiters with Lugol's solution, we are in agreement with the results published by Rienhoff and various other authors. Our results are also in agreement with the experiments on animals carried out by Breitner and also with the rule advanced by Breitner¹ that the effect of the iodine ingested takes place in the opposite direction to the functional direction of the thyroid gland.

As is well known there does not exist any homogeneous pathognomonic, anatomic structure of *secondary* toxic goiter (toxic adenoma, secondary Basedow disease). The histological appearance of these goiters may vary to a large extent both with regard to the cells and the colloid. We may also frequently find varying pictures in the same secondary toxic goiter, nodes rich and poor in colloid

¹ BREITNER, D. B. The iodine question in animal experiments. AM. J. SURG., 6: 17, 1929.

* Studies from the Surgical Clinic A (Medical officer: Prof. Dr. Johan Nicolaysen), and the Pharmacological Institute (Prof. Dr. E. Poulsson) of the University of Oslo, Norway. Read at the Annual Meeting of the American Association for the Study of Goiter, Denver, June 18-20, 1928, by Gulbrand Lunde.

unevenly distributed and it is quite impossible from their histological construction to state whether they are secondary toxic goiters or not. Owing to these facts it is difficult or even impossible to determine the effect of the iodine treatment on the histological picture of secondary toxic goiter. Nevertheless it may be mentioned that in all the secondary toxic goiters studied by us we found much colloid after the iodine treatment.

IODINE CONTENT OF UNTREATED TOXIC GOITERS

In Table 1 we have set up the determinations of the iodine content of 4 untreated toxic goiters and the results of the histological investigations compared with the metabolism figures of the respective patients and the iodine content of normal Norwegian thyroid glands.

TABLE I
IODINE CONTENT OF THYROIDS (MG. PER CENT) OF FRESH
(MOIST) TISSUE

	Mg. Per Cent
Untreated primary toxic goiters.....	<div style="display: inline-block; vertical-align: middle;"> <div style="font-size: 2em; vertical-align: middle; margin-right: 5px;">{</div> <div style="display: inline-block; vertical-align: middle;"> <div style="text-align: right; padding-right: 5px;">4.14</div> <div style="text-align: right; padding-right: 5px;">8.8</div> </div> </div>
Untreated secondary toxic goiters.....	<div style="display: inline-block; vertical-align: middle;"> <div style="font-size: 2em; vertical-align: middle; margin-right: 5px;">{</div> <div style="display: inline-block; vertical-align: middle;"> <div style="text-align: right; padding-right: 5px;">20.0</div> <div style="text-align: right; padding-right: 5px;">122.9</div> </div> </div>
Normal Norwegian thyroids.....	<div style="display: inline-block; vertical-align: middle;"> <div style="font-size: 2em; vertical-align: middle; margin-right: 5px;">{</div> <div style="display: inline-block; vertical-align: middle;"> <div style="text-align: right; padding-right: 5px;">28.2*</div> <div style="text-align: right; padding-right: 5px;">46.2</div> <div style="text-align: right; padding-right: 5px;">56.5</div> <div style="text-align: right; padding-right: 5px;">102.0</div> </div> </div>

* This thyroid weighed 35 gm.

The first 2 goiters in Table 1, as regards both their clinical symptoms and their pathological, anatomical pictures are typical primary toxic goiters (genuine exophthalmic goiters) with extremely little colloid. The second 2 goiters must be regarded as secondary toxic goiters rich in colloid (secondary exophthalmic goiter, toxic adenoma).

The iodine content determined in the 2 cases of primary toxic goiter is seen to be very low, and here we are in agreement with previous investigations. Especially when compared with the iodine content of normal Norwegian thyroid glands, the iodine poverty becomes striking.

One of these thyroids weighs 35 gm. which is so high that we can scarcely regard this gland as normal. In our opinion a normal thyroid gland in Norway ought not to weigh more than 30 gm. The thyroid weighing 35 gm. was also from an inland district where some goiter is to be found. If we leave this thyroid out of account we can compute the average iodine content of the Norwegian thyroids investigated, and we find that it varies from 50 to 100 mg. per cent. These figures are much higher than in any other land where such investigations have been carried out. This is probably caused by the fact that much sea-fish is used as food, especially in our coast districts.

If we now look at the 2 secondary toxic goiters in Table 1 we find quite different facts than in the case of the primary goiters, as regards both the colloid and the iodine content. The secondary toxic goiters are rich in colloid and rich in iodine. One of these shown in Table 1 is even considerably richer in iodine than any of the normal thyroids investigated. How two types of goiter showing so different qualities can produce the same disease, exophthalmic goiter, is a question which we cannot discuss here. We will only call attention to the fact that as far as their colloid and iodine content are concerned, the two secondary toxic goiters examined by us, that had never been treated with iodine, showed qualities similar to the goiters from patients with definite medicamental iodine-thyrotoxicosis, examined by Jansen and Robert. One of us (Holst) has previously suggested that some of the secondary thyrotoxicoses in Norway may have been produced in the following way: The individual may have spent his infancy and youth in an iodine-deficient district, and been brought up on the local iodine-deficient food. On account of this he developed a node-goiter (struma nodosa). In later life he may have changed the character of his food to foodstuffs more rich in iodine, and on account of this the goiter may have become secondary toxic. The iodine

content of the two secondary toxic goiters examined by us probably supports this assumption.

IODINE CONTENT IN TREATED CASES

We now come to the results of our investigations on the *preoperative iodine treatment* by the method of Plummer by primary thyrotoxicosis (genuine exophthalmic goiter). The results of the histological and chemical investigations on 7 primary toxic goiters removed after having finished a complete and effective treatment with Lugol's solution showed them all to be rich in colloid and to contain iodine: 19.9, 22.0, 33.0, 39.4, 43.7, 64.7, and 64.9 mg. per cent respectively.

From these figures we may conclude that *these goiters have changed during the iodine treatment from being poor in colloid and iodine into goiters rich in colloid and iodine.*

RELATION OF COLLOID AND IODINE IN GOITERS TO BLOOD IODINE

We will now try to make clear the manner in which the changes in the colloid and iodine content of the primary toxic goiters already mentioned are related to changes in the blood iodine.

Gley and Bourcet were the first to assert that iodine was a normal component of blood. Blum and Grützner, however, denied the existence of iodine in the blood, if food containing no iodine was used. A sufficient answer to this assertion is a reference to the fact that it is quite impossible to produce a food which contains no iodine. On the other hand the method applied by these authors was not sensitive enough to detect and determine quantitatively the small amounts of iodine in the blood. Later investigations carried out by Kendall and Richardson confirmed the results obtained by Gley and Bourcet. They determined the average physiological figure for the blood iodine at 13 gm. per cent. Veil and Sturm determined the normal iodine content in Munich to 12.8 gm. per cent in the autumn and 8.3 gm. per cent in winter. Mauer and Dietz found 4.2 to

15 gm. per cent (average 9.2 gm. per cent). Jansen and Roberts 10–16.2 gm. per cent (average 12 gm. per cent). We determined the normal iodine content of blood in Oslo in 2 cases at 11 gm. per cent and 16 gm. per cent.

Von Fellingerg showed that the blood iodine might be divided into an organic and an inorganic component. He separated the two components so that the blood was coagulated in alcohol and the coagulum was then washed out several times with alcohol. By this method the inorganic iodine will be dissolved in the alcohol. Veil and Sturm also used this method in their experiments. The method is however very elaborate and takes much time. Veil and Sturm themselves report that it was necessary to wash the coagulum up to ten times before it was free of inorganic iodine. In collaboration with Karl Closs one of us (Lunde) worked out another method that was used for the determinations of blood iodine as reported in this paper.

We shall briefly describe the method: five to 10 c.cm. of blood are taken out with a syringe which is first washed with a citrate solution. The blood is immediately injected into a flask containing about four times as much of 96 per cent iodine-free alcohol, and is thoroughly shaken. The finely divided coagulum is now put into a Soxhlet apparatus and is extracted with alcohol for four hours. The whole inorganic iodine is now dissolved in the alcohol and the compounds which are indissoluble in alcohol left. The iodine may now be determined separately in each of these fractions. As to the iodine determination itself we may refer to previous papers published by Gulbrand Lunde and collaborators.

The results of the investigations hitherto carried out by us are that in all the cases examined we were able to detect and quantitatively determine the iodine content of the blood. Our results are thus in agreement with those of the previous investigations above mentioned. *Iodine is a normal component of blood.* In all the cases

examined we were able to determine the inorganic as well as the organic component of the blood iodine, except in a few cases of hyperthyroidism in which the organic component insoluble in alcohol was reduced, by means of ingested inorganic iodine, to such a small amount that it could not be detected.

We shall now see what we can discover in regard to the different iodine compounds present in the blood. We divided the alcohol-soluble iodine compounds in the following way: first the alcoholic extract was evaporated nearly to dryness after that a few drops of potassium iodide had been added. The residue was dissolved in water, acidified and shaken out with chloroform. The two layers were then separated by means of a separating funnel in a centrifuge. In the chloroform was dissolved the total iodine combined with fats or lipoids. We have called this fraction the lipid-iodine fraction. In a great number of investigations we determined this lipid-iodine, but the quantity determined was in every case so small that it could be disregarded.

If chloroform and some nitrite-sulphuric acid is added to the remaining aqueous solution, the inorganic iodine will be set free and may be shaken out with the chloroform. Our experiments, however, showed that the total inorganic iodine could not always be found in this way. We are of the opinion that this is due to the fact that the iodides are partly oxidized to iodate and not to iodine alone. This iodate cannot be shaken out with chloroform, and the figures for the inorganic component of the blood iodine will be too small. At present we are working out another method which we hope will in all cases give up quite correct figures for the inorganic component.

After we have removed these inorganic compounds, there will still remain iodine in organic combination in the aqueous solution. This latter fraction of the blood iodine has been overlooked by previous

investigators. Veil and Sturm, for instance, regard all the iodine which remains after the lipid fraction has been removed as inorganic. Therefore when Veil and Sturm give the average for the total iodine present in normal blood in organic combination as 65 per cent, we may be assured that this figure is too low, and here we are quite in agreement with von Fellenberg.

We now come to the blood that remains in the coagulum after the extraction with alcohol in the Soxhlet apparatus. As this fraction (insoluble in alcohol) is inseparably combined with the albumens which are coagulable with alcohol, it may probably consist of albumen iodine. In a few cases examined by us we determined this alcohol-insoluble fraction at 1 to 47 per cent. The results of previous investigations (Oswald, Kendall) compared with our own results have led us to the opinion that this fraction must contain the active principle of the thyroid secretion. The active iodine-containing substances in the thyroid gland are just such albumen compounds.

Owing to these facts it was of the greatest importance to determine this albumen-iodine fraction in our cases of thyrotoxicosis. Veil and Sturm found that in cases of thyrotoxicosis there were always increased figures for the blood iodine. As will be seen from Figure 1, *we always found increased figures not only for the total blood iodine, but also very high figures for the alcohol-insoluble fraction.*

As we might expect, the alcohol-soluble fraction (A) of the blood iodine increases considerably on account of the iodine overflow after the treatment with inorganic iodine, whereas the alcohol-insoluble fraction (B) is reduced to the normal or almost to the normal figures, and the fall is practically parallel to the decrease of the metabolism figures during the treatment with Lugol's solution.

We desire to call attention to the following facts:

1. On account of the chemical qualities displayed we must assume that the fraction

B contains a considerable amount of the products secreted from the thyroid gland and circulating in the blood.

2. We find that the fraction B is increased in all the cases of untreated primary thyrotoxicosis.

3. We further find that the fraction B falls to the normal practically parallel to the decrease of the metabolism figures, and at the same time that the patient becomes free of symptoms.

From these facts we may conclude that the fraction B at least partly contains the active thyroid products circulating in the blood. If this holds true the increase of the fraction B must be an essential cause of thyrotoxicosis.

There was an increase of the fraction B in all the cases of untreated primary thyrotoxicosis examined. No direct proportion, however, between the figures of the fraction B and of the metabolism can be stated. The parallelism between the curves of the fraction B and the metabolism figures is also incomplete. In some cases we find small deviations between the curve of metabolism and that of the fraction B. Fraction B cannot therefore be regarded as a measure for the degree of the thyrotoxicosis. And this could hardly have been expected, for the fraction B may possibly contain other iodine compounds which are not derived from the thyroid gland. Veil and Sturm showed for instance that organic iodine compounds may be reabsorbed from the intestinal canal and circulate as organic compounds in the blood.

The blood samples examined by us were in all cases taken from fasting patients that in no case had eaten any food especially rich in iodine (for instance sea-fish) during the two last days. It is even not impossible that organic compounds derived from food may have found their way into the fraction B.

On the other hand, iodine compounds derived from the thyroid gland may probably have found their way into fraction A and appeared among the alcohol-soluble organic compounds. These two facts may be the reason why we do not find

a complete agreement between the curves for the fraction B and for the metabolism.

SUMMARY

The most important results of our investigations may be illustrated and summarized as follows:

1. In the case of untreated primary thyrotoxicosis the figures for metabolism and for both fractions A and B of the blood iodine are increased. The goiter is poor in colloid and iodine.

2. Through treatment with Lugol's solution an enormous and quite abnormal increase of the inorganic component of the blood iodine is produced. Influenced by these large amounts of inorganic iodine in the blood, the fraction B is forced down to the normal, and at the same time a retention of the colloid and iodine in the goiter takes place.

3. We can understand these facts as demonstrating that large amounts of inorganic iodine in the blood constipate the secretion from the thyroid gland into the blood, and increase the ability of the gland to store up the secretion. As a consequence, the blood is no longer flooded with thyroid secretion. This is made clear by the fact that the fraction B sinks towards the normal, and an improvement takes place in the thyrotoxicosis as demonstrated by the change in the clinical symptoms and the metabolism figures.

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UTAH GOITER SURVEY*

INCLUDING EXAMINATION OF 110,000 CHILDREN

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FOR many years the Utah State Board of Health has been interested in the goiter problem. It attempted limited surveys as early as 1910, but it was not until 1924 that it was possible to make a state-wide survey, which was deemed advisable before undertaking any prophylactic measures.

Dr. James Wallace was placed at the head of the work in 1924 and the examinations were completed in 1925. School children to the number of 90,000 were examined by Dr. Wallace and the remaining 20,000 under his supervision. A preliminary article, covering the first 69,000 children, was published.¹

The complete report of the Utah investigation is quite lengthy and cannot be given in detail here. At best, we can briefly discuss the general considerations involved.

Utah contains 84,990 square miles, an area one and one-half times that of Michigan. The goiter survey is the largest numerically and the most extensive geographically of any in the United States. The total number of school children examined was 110,086, 54,935 boys and 55,151 girls, representing approximately 25 per cent of the total population of the state.

The examination was conducted by inspection of each pupil in a good light. The thyroid gland was palpated and again palpated as the pupil swallowed. In these examinations, the cooperation of the local doctors was sought and in some of the larger centers many of the examinations were made under supervision.

An attempt was made to standardize the diagnosis as much as possible in order that the findings of the different examiners might be on a uniform basis and therefore comparable.

Four degrees of enlargement were recorded: (1) "The question mark or pre-goitrous group," where the neck could not be pronounced negative and yet the degree of enlargement was so slight that one might hesitate to say the individual had goiter; (2) "slight," any enlargement up to 1 in.; (3) "moderate," any enlargement from 1 to 2 in.; (4) "great," any enlargement over 2 in.

The survey showed the average goiter incidence to be 32 per cent. among the boys and 54.5 per cent among the girls. A dozen localities showed less than 10 per cent goiter and in two or three of these communities almost every case was an imported one. In contrast to these places there were sections where every boy and girl in school showed some degree of enlargement. Again, in one district, there was a variation from 6 per cent in one school to 100 per cent in another near by. But these schools were situated differently from a geological standpoint.

Furthermore, no racial immunity to thyroid enlargement was found. Representatives of practically every race that immigrates to America were examined, in one school thirty-six different nationalities. Interest was centered on the Indians because of an opportunity afforded to examine a considerable number of these people living, as far as water and food supply are concerned, under conditions similar to the local whites. Indians, as a rule, were found to be slightly less susceptible to goiter. It was noted that male Indians had a higher incidence of thyroid enlargement than females. This peculiarity was found to be true of the Indians whether on reservations or in the public schools.

Samples of drinking water from many sections of the state were examined by Dr. J. F. McClendon of the University of

¹ Wallace, J. Goiter survey in Utah. *Calif. & West. Med.* 22: 431, 1924.

*Read at the Annual Meeting of the American Association for the Study of Goiter, Denver, June 18-20, 1928.

Minnesota. His results indicate a less definite inverse relationship between iodine in the drinking water and the occurrence of goiter than figures from other parts of the country. With the exception of the Milford sample they all showed marked iodine deficiency, when judged by the standard that proper drinking water supply required five parts per billion of iodine.

A sample of water from Great Salt Lake, the principle source of the table salt used in Utah, yielded seventy parts of iodine per billion of water. All of this iodine is lost in the process of manufacturing table salt on account of the method used in precipitation. The following striking fact was noted in Kaysville, a town on the shore of Great Salt Lake: Formerly the citizens produced their table salt locally by a process of evaporation without refining. The incidence of goiter at that time was very low. Later, when the local method of production was abandoned and the commercial product substituted, there was a large noticeable increase of goiter. A like increase in several localities has followed the installation of improved water supplies from mountain sources replacing local wells.

ADULT SURVEYS

In order to determine approximately in what percentage of children thyroid enlargement was "purely physiological and would naturally recede," it was decided to make a survey of adults and see how they compared with the school children. Six towns were selected, four in the south, one about the center of population and one in the north. These towns were surveyed by a house to house visitation, in order that every adult would be included; and only half a dozen persons refused examination.

Of these localities, Farmington, Oak City, Providence and Cedar City were chosen because a high percentage of goiter had been found among the school population; Delta and Milford, however, were chosen because the incidence was low. These two towns are comparatively new.

Details were taken regarding the length of residence in the town of the adults examined, and the comparative freedom from thyroid enlargement among those who had lived in the region for some time. The water supply of this locality is from wells 200 to 500 feet deep.

The details gathered further showed that in a goitrous area like Utah there is very little less enlargement of the thyroid gland among the adult population than among the school population and that the number of cases that are only "temporary" in children is very small and that of all those who develop enlargements when children, not more than about 20 per cent of these enlargements, among the males and 10 per cent among the females, disappear spontaneously.

PROPHYLAXIS

A large amount of prophylactic work has been done in the schools. The State Board of Health has urged the use of chocolate tablets containing 10 mg. of iodine, one tablet to be taken once a week for the forty weeks of the school year. Through the cooperation of the superintendents and teachers, in one year, more than 1,200,000 of these tablets were administered to the pupils. Many more have been administered which have not been reported to the State Board of Health. Besides this many parents, following the ideal method, have taken their children to their own physicians for prophylaxis or treatment.

In the foregoing summary of the Utah goiter survey it has been pointed out that the thyroid gland enlargement in children, among all races of people, may be found in all sections of the state, with marked local variations of incidence and that the occurrence of goiter among adults is not much less than among children. It has also been shown that there is a lack of correlation between the percentage of iodine in the samples of drinking water from different localities and in the prevalence of goiter in these same communities.

There is another interesting aspect of the Utah problem which was not covered in

any detail in the State survey. It has to do with the geological features of the intermountain region, which differ from every other section of the United States. Practically the whole of America is drained by water sheds which discharge their burdens into streams which flow into the ocean. In the arid region between the Uinta mountains on the east and the Sierra Nevada mountains on the west involving the western half of Utah, small contiguous sections of Wyoming, Idaho and Oregon, nearly all of Nevada, a large part of eastern and southern California and extending into Mexico, is a land area called the Great Basin which has no outlet to the ocean. All the streams of this region flow into saline lakes or evaporate.

In the Quaternary geological period, Canada and the northern half of the United States were covered by four million square miles of ice, during the so-called Ice Period. This sheet did not cover the section west of the Rocky Mountains. At that time the precipitation was much more abundant than at present. There was more water falling than was evaporating and it gathered in the various depressions in the bottom of the Great Basin, forming numerous lakes of which we still have evidence. In Utah this water accumulated to the depth of a thousand feet and formed a lake called Lake Bonneville, the size of Lake Michigan, at which point it stayed for a long period, etching into the mountain sides a line of wave-wrought cliffs and depositing under the water edge the disintegrated rock in the form of a level shelf of sand and gravel. That this level was maintained for a long time is evidenced by the fact that this underwater shelf had time to build itself several hundred yards wide in places. This level known as the Bonneville Shore Line persists to the present day and can be traced along the mountain sides for hundreds of miles.

The outlet of this lake was at the north and was through a rather loose formation through which the discharging waters cut

a channel, four hundred feet deep, to be halted by a hard limestone ledge which held the waters at this stage long enough to build another definitely defined shoreline which is called the Provo Shore Line. At this time precipitation diminished to the point where it was insufficient to keep the lake full. Then the water receded rapidly and left two remnants of Lake Bonneville, Great Salt Lake on the north and Sevier Lake on the south.

At the point where the lake failed to empty itself the character of the water began to change; the soluble minerals from its drainage area were brought down to the lake and remained in solution. Through the ages those minerals became more concentrated as there were more coming in each year and the lake water was getting smaller in amount. This process did not leave its mark until the dessication and concentration reached the lower levels of the lake bottom and then the chemicals were deposited. This process is still going on.

Great Salt Lake, during the last seventy-five years, has varied as much as 9 feet in its average depth, because of the irregularity of seasonal precipitation and evaporation. At its low point it has left great stretches of richly mineralized deposits on the shallow at the edge of the lake. The waters of the lake itself contain 15 to 18 per cent sodium chloride, according to the season; 2 per cent phosphates; numerous other chemicals in smaller amounts and seventy parts per billion of iodine, a higher percentage than any of the ocean analyses.

Sevier Lake, the southern remnant of Lake Bonneville, is going through the same fluctuations as Great Salt Lake and has left, during its desiccation and shrinkage, a flat desert country to the north richly impregnated with minerals which in agriculture are designated under the generic term "alkali" which indicates all those chemicals which in sufficient concentration prevent the growth of vegetation.

In order that we may study the possible relationship of the Lake Bonneville phenomenon to the distribution of goiter, we

may glance at a map of the State of Utah and see that the mountainous regions on and above the Bonneville shore lines show a high percentage of goiter, while the towns in the desert country, representing the lowest part of the old lake bottom, show a low percentage. A map of Millard county illustrates these sharply contrasting conditions within a small area; therefore we shall use this county for illustration because it has been twice surveyed in detail, by the State Board of Health in 1924 and by the writer in 1923.

On the east side of Millard County, situated on the level bench land above the Provo shore line, are six old mountain towns, Leamington, Oak City, Holden, Fillmore, Meadow and Kanosh, which show an average of 73 per cent goiter among the school children. The drinking water supply of this group is derived from the adjacent mountains and is low in iodine. Oak City had 0.017 parts of iodine per billion. Her goiter percentage in 1923 was 90 and in 1924, after a year of prophylaxis, it was 82. The people of this district live largely on the locally grown produce, cultivated on the soil washed down from the same mountains which furnish the drinking and irrigation water. Here the problem seems to be comparatively simple because analyses of the drinking water give the clue to the total iodine as represented in drinking water, irrigation water, soil and food.

Twenty to forty miles away, on the lake bottom desert is another group of old towns, Hinckley, Oasis and Desert, which have an average of about 10 per cent goiter. Near them is the town of Delta which is comparatively new and offers an opportunity to contrast the old inhabitants with the new, to show what effect desert dwelling has upon those recruited largely from the goiter districts.

The school figures of Delta showed 22 per cent thyroid enlargement. A residence duration survey of the pupils showed that children living there five years or longer had only 11 per cent goiter.

The ground waters of this area have been thoroughly studied by the United States Geological Survey. The drinking water is obtained from artesian wells which tap several water bearing strata at different depths. These strata are supplied from a catchment area above the Provo shore line twenty to forty miles away. This accounts for the fact that the drinking water analysis from two artesian levels showed .029 and .08 parts per billion of iodine. It is immediately apparent that the amount of iodine in the drinking water does not explain the fact that there is a small amount of goiter on the desert and a large amount in the nearby mountains.

The irrigation water in the desert is entirely different from the drinking water. It comes from the Sevier River which travels 300 miles through all sorts of formations before being spread upon the land. In looking for another source of iodine a chemical investigation will be pursued until the rôle of irrigation water is understood.

Soil is the next possible factor influencing goiter. Fifteen hundred soil samples from this section have been analyzed in detail, with the exception of iodine determination. The local station of the Department of Agriculture is now prepared to investigate iodine. This promises to be an extremely interesting study because of the high alkali content of the soils.

Food analyses have not been made with the exception of a few specimens of wheat from Oak City examined by Forbes and his associates of the Ohio Experiment Station. A trace of iodine was found in 1 sample.

This brief discussion of the Lake Bonneville relationship to goiter is in the nature of a preliminary report only. There seems to be sufficient evidence to justify the hypothesis that: The soil and irrigation water as contributors to the food chemistry may be the sources of sufficient additional iodine to account for the small amount of goiter in the desert country. At the completion of the irrigation and soil analysis a full report will be made.

SIGNIFICANCE OF LOW BASAL METABOLISM FOLLOWING THYROTOXICOSIS*

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BOSTON

DURING the period 1915 to 1926 approximately 487 cases of toxic (most exophthalmic) goiter were treated by surgery, by roentgen ray, or by iodine alone, at the Massachusetts General Hospital. About half of these were followed for one to eleven years after treatment. Among the cases thus followed 66 had a basal metabolism below minus 15 per cent after treatment. Of these 66 patients, 11 had signs and symptoms that were characteristic of myxedema.† Seven others temporarily presented a syndrome somewhat suggestive of myxedema, but later were symptom-free with a low metabolism. The majority of the patients, however, showed no evidence of thyroid underfunction and appeared to be healthy individuals at the time of their low metabolism.

I. LOW BASAL METABOLISM WITH MYXEDEMA, FOLLOWING THYROTOXICOSIS

The basal metabolic rate ranged from minus 20 to minus 37 per cent.

One of the most significant things revealed by this study was the rarity of definite myxedema following all forms of therapy for toxic goiter.¹ Its incidence was only a little over 2 per cent. There is 1 case of myxedema following untreated

† The number of cases given in an earlier report on myxedema following thyrotoxicosis¹ has been augmented by one case originally classed as temporary low metabolism without myxedema (Lab. no. 3543, Case 5 in Ref. 2). We have recently observed typical myxedema twice in this patient, each time during the postoperative administration of iodine, making it appear highly probable that her low metabolism which occurred immediately after thyroidectomy, when she was taking iodine, was due to hypothyroidism which did not last long enough to become clinically detectable. This explains why the number of cases of temporary low metabolism without myxedema has been reduced from 27 in the earlier report² to 26 in this.

* Read in abstract at the Annual Meeting of the American Association for the Study of Goiter, Denver, June 20, 1928. This study was aided in part by a grant from the Proctor Fund of the Harvard Medical School, for the Study of Chronic Diseases. From the Thyroid Clinic and Metabolism Laboratory of the Massachusetts General Hospital.

toxic goiter on record in this clinic. Inasmuch as the incidence of myxedema is so low following treatment, this case emphasizes the importance of determining how frequently it follows untreated toxic goiter before ascribing its occurrence to any form of therapy. At present this is unknown.

In 3 of the 11 patients with definite myxedema, the disease was of only temporary duration.‡ In the other 8 cases it appeared to be permanent, although the permanency has not been tested in every instance by discontinuing the administration of desiccated thyroid.

The data are not extensive enough to warrant a definite statement, but the incidence of the permanent type appeared to be slightly greater following roentgen-ray therapy (about 4 per cent) than following subtotal thyroidectomy (about 1 per cent).

A feature of the cases following roentgen-ray therapy was the late onset of the myxedema after treatment (two to five years) whereas, in the cases following subtotal thyroidectomy it occurred within a few months after treatment.

II. LOW BASAL METABOLISM WITHOUT MYXEDEMA FOLLOWING THYROTOXICOSIS

The remaining 55 patients had a low basal metabolism without definite signs and symptoms of underfunction of the thyroid. In 26 of these the low metabolism lasted only for a time,² whereas in 21

‡ Seven cases in the non-myxedematous group temporarily showed puffiness of the face without sufficient accompanying signs and symptoms to warrant a definite diagnosis of temporary myxedema. This phenomenon occurred with a normal as well as with a subnormal metabolism. Until its nature is definitely established, we prefer to refer it as a temporary edema and not as temporary myxedema.³

it appeared to be relatively permanent.⁴ These have been called temporary and permanent types respectively. They will

to standard normal while the patient was still on iodine, and those in which it rose to standard normal only when iodine was

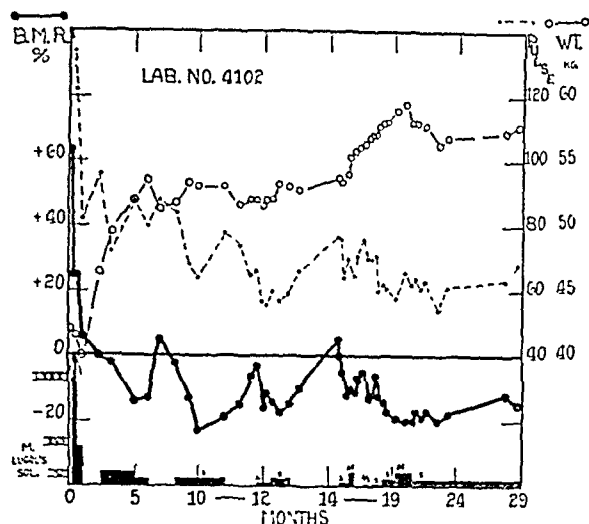


FIG. 1. Mrs. N. L., aged twenty-three years. Repeated production of temporary low basal metabolism, coincident with the administration and omission of iodine, following subtotal thyroidectomy (arrow) for exophthalmic goiter. Nervousness decreased with each metabolic depression. No myxedema at any time.

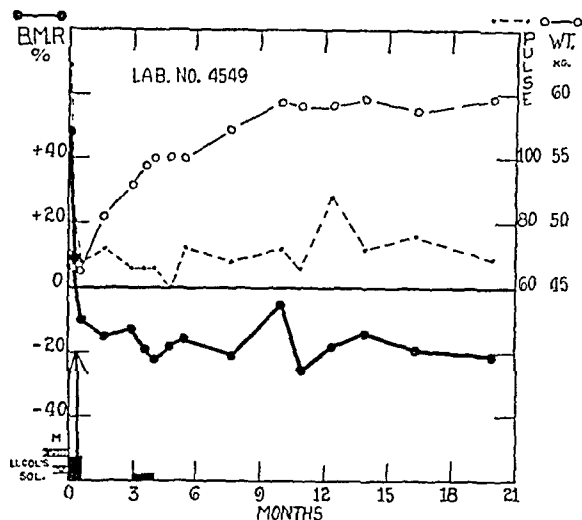


FIG. 2. Mrs. A. G., aged forty-two years. Permanent low basal metabolism without myxedema, following subtotal thyroidectomy (arrow) for exophthalmic goiter. The metabolism remained low after omission of iodine, in contrast to what happens in cases of temporary low metabolism, as shown in Figure 1.

be dealt with separately, but a probable relationship that exists between many of them will be mentioned in the discussion. It was impossible to classify the remaining 8 patients with regard to the duration of their low metabolism.

Temporary Type. The basal metabolism ranged from minus 16 to minus 44 per cent.

The time of onset of the temporary low metabolism ranged from almost immediately after treatment to several years later, but most of the cases appeared during the first four months of convalescence, and about half of these within the first month.

In the majority of instances the low metabolism did not last longer than one to four months.

Although 5 cases had no iodine at or near the time of the low metabolic rate, in most instances the patients were receiving Lugol's solution during this period. In the latter group there were two types of cases, viz., those in which the metabolism rose

omitted. The majority of the iodine cases showed the latter phenomenon, and in some of them it has been repeated several times by a similar manipulation of iodine therapy. (See Fig. 1.) In these cases it appeared possible to maintain the low metabolism indefinitely by continuing the administration of iodine.

In 1 of the patients there were signs and symptoms suggestive of myxedema at the time of the first period of low metabolism, but no suggestion of myxedema at the time of her subsequent five periods of low metabolism, one of which lasted for six months.

It is of some interest that in a few cases the low metabolism occurred during treatment by iodine alone, without previous surgical or roentgen-ray therapy.

Permanent Type. There were 21 patients who, eventually, without clinical evidence of myxedema, had a low metabolic rate, presumably permanent, ranging from minus 16 to minus 25 per cent, after recovery from thyrotoxicosis.⁴ Among these patients there were 6 who shortly after treatment had transient signs and

symptoms which made it necessary to consider the presence of myxedema. Later on, in spite of no change in metabolism, these signs and symptoms disappeared and did not recur.

In the cases where it was possible to make an estimate, the time of onset varied from three months to over one year after the completion of treatment.

Data on a typical case are plotted in Figure 2.

These patients with permanent low metabolism without myxedema fall into two general groups:

1. Those who appeared in other respects to be normal individuals, 15 cases.

2. Those who appeared to be somewhat abnormal, 6 cases.

The most common complaints of the second group were nervousness, ease of fatigue and sensitiveness to cold. Their skin was soft and warm, the hair was usually not dry and there was no edema. The administration of desiccated thyroid sometimes made them tire a little less easily without necessarily producing much effect on the metabolic rate.

In 3 cases an artificial menopause, and in 1 case a spontaneous menopause, occurred either before or after treatment, but the data are of such a nature that the influence of this factor cannot be determined.

DISCUSSION

In most of the cases of low metabolism with myxedema the rate was below minus 25 per cent. In most of the cases of low metabolism without myxedema the rate was above minus 25 per cent. We have not seen an individual with a basal metabolism below minus 25 per cent that could be regarded as healthy. We have seen several cases of well marked myxedema with a metabolic rate above minus 25 per cent. Most reductions of the basal metabolic rate to a level below minus 25 per cent appear to be due to an underfunction of the thyroid. Most basal metabolic rates above this level apparently are not. These

statements apply not only to low metabolism following thyrotoxicosis, but also to low metabolism in general as observed at this hospital.

Our reasons for assuming that the majority of our patients with permanent low metabolism following thyrotoxicosis did not have myxedema are:

1. During the period of their low metabolism (regardless of its duration) the majority of them seemed to be active healthy individuals.

2. The lack of clinical benefit in most instances following the administration of desiccated thyroid.

It is, of course, already known that there are several pathological entities other than myxedema, such as starvation, chronic nephritis and underfunction of various endocrine glands, that are associated with a low metabolism in some instances.* The only one of these that was a factor in our series was hypogonadism (4 cases). There is, however, no conclusive evidence in the literature that hypogonadism per se is a cause of low metabolism. It seems to us highly probable that there are still other obscure pathological entities about which nothing is known at present, that are associated with a low metabolic rate. It may be that some of our abnormal cases fall into this category.

This would leave, however, a large number of patients with a low metabolism entirely unaccounted for. These individuals appear active and healthy and able to function in a manner that is sufficiently satisfactory to be classed as normal. It is probable that metabolic standards are like height-weight tables, and that there

* Means and Burgess⁵ in a survey of the first 1000 patients on whom basal metabolism tests were done in this clinic, found 63 who were not frankly myxedematous or cretinoid, in whom the basal metabolism ranged from minus 11 to minus 21 per cent. They classified these 63 patients as follows:

Suspected hyperthyroidism	12
Non-toxic goiter	6
Miscellaneous non-endocrine.	8
Suspected hypothyroidism	15
Other endocrines	20
Blood diseases	2

are frequent deviations from the average that are not necessarily abnormal. To try to fit metabolic standards into too narrow ical differences in patients themselves. Several of our cases in which thyroid had no clinical effect were young people who

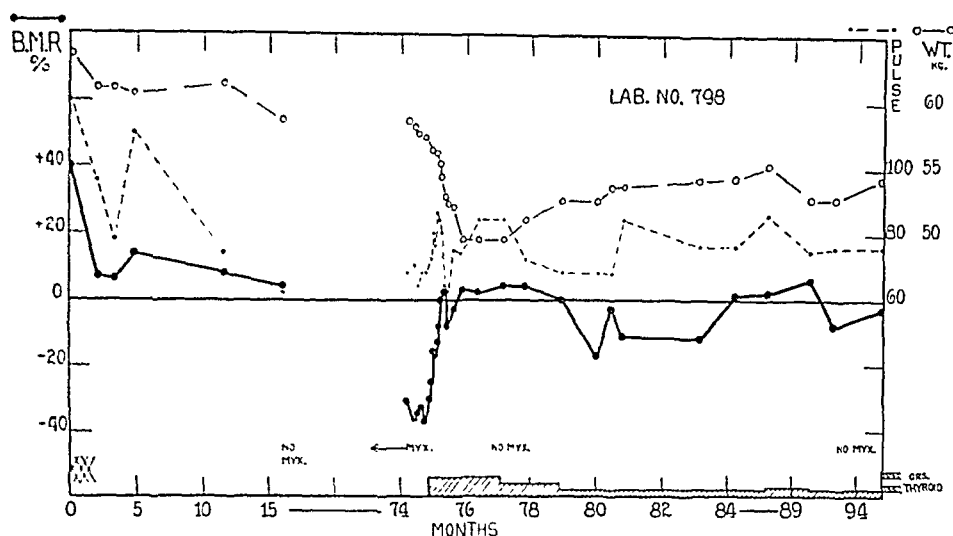


FIG. 3. Miss A. K. R., aged seventeen years. Showing the prompt response of the basal metabolism to small doses of thyroid (Armour's, 3 grains daily) in a typical case of myxedema following roentgen-ray therapy (X) for exophthalmic goiter. The administration of $1\frac{1}{2}$ grains daily appeared sufficient to maintain the metabolism at a standard normal level indefinitely.

a groove is to ignore the fundamental biological concept of variation.*

For the past few years it has been noted by several observers that doses of desiccated thyroid that would be more than ample for patients with myxedema would have little effect on some of their cases of low metabolism without myxedema. Nevertheless, the low metabolism has usually been considered due to an underfunction of the thyroid, directly or indirectly, and the condition is frequently referred to as "hypothyroidism without myxedema." The explanations offered for the lack of effect of thyroid preparations in these instances indicate the current attitude towards the status of the patient. It is ascribed to such various causes as the longstanding nature of the symptoms with complicating arteriosclerosis,⁷ lack of absorption from the gastrointestinal tract,⁸ loss of potency of the thyroid preparation or insufficiency of dosage.^{8,9} Practically no consideration is given to possible biolog-

* It is of considerable interest that Du Bois has recently decided that the Aub-Du Bois standards (used throughout in our calculations) are 6 per cent too high.⁶

did not have any arteriosclerotic changes. In many others we are sure that potency, absorption and dosage were not at fault, because the metabolism rose to standard normal, whether with varying doses of desiccated thyroid by mouth or with intravenous thyroxin, yet there was no improvement clinically. That is to say, we got metabolic but not clinical results. In some instances where we did not get even a significant effect on metabolism with ordinary doses, we are sure that the desiccated thyroid was absorbed, because symptoms of thyroid intoxication supervened whenever the dose was increased beyond the patient's tolerance, even though the metabolic rate remained distinctly subnormal.⁴ (See Fig. 4.)

Some patients with low metabolism and no myxedema respond readily to moderate doses of desiccated thyroid with a well-marked rise in metabolism, without necessarily showing any clinical improvement. It is more common, however, for them to require large doses to affect the metabolism perceptibly. A similar difficulty is often experienced in trying to raise the meta-

bolism of patients whose normal rate is at the standard level. In other words, the reaction to thyroid therapy in low rate

normal. There was one exception to this, however. In this patient typical myxedema was observed twice, each time during the

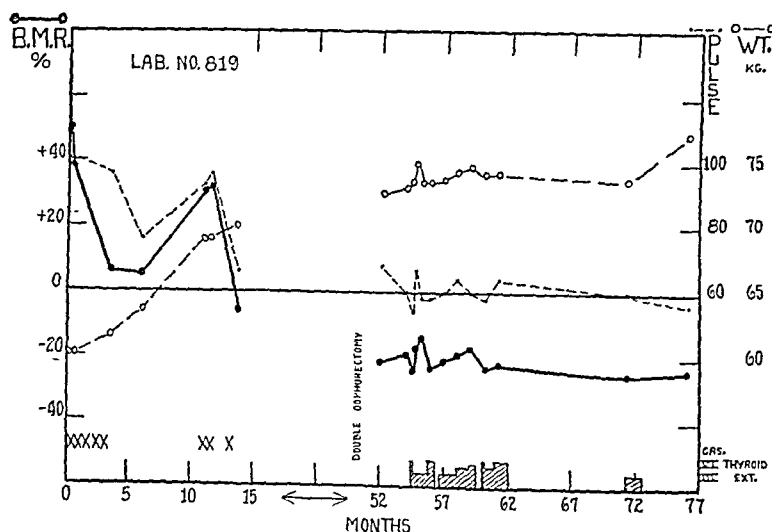


FIG. 4. Mrs. A. A., aged forty-four years. Showing practically no effect on basal metabolism of 6 grains of thyroid (B. W. & Co.) daily in a patient with permanent low metabolism following roentgen-ray treatment (X) for exophthalmic goiter. Although the thyroid administered did not significantly affect the metabolism, it did cause symptoms of thyroid intoxication.

cases without myxedema is often precisely what one would expect it to be provided the low metabolism be normal for them.

As a general rule, the patient who requires more than 4 grains of desiccated thyroid (Armour's) to raise the metabolism to standard normal has either not got myxedema, or else if he has that deficiency, has in addition a low rate normally. The maintenance dose for most cases of true myxedema seen in this clinic is $1\frac{1}{2}$ to 3 grains of desiccated thyroid (Armour's) daily.

The prompt response of the metabolism to small doses of thyroid is shown in a typical case of myxedema in Figure 3 and may be contrasted with the comparative lack of effect from the same or larger doses in a low rate case without myxedema, as shown in Figure 4.

The explanation of temporary low metabolism is more difficult. In the cases where the rate could be held at a low level indefinitely by the postoperative administration of iodine, there was usually no evidence of myxedema and the patients often felt better than when their rate was standard

postoperative administration of iodine. Plummer¹⁰ and Haines¹¹ have reported a similar case and Plummer¹² states that he has seen a slight depression of the metabolism occur in a few normal individuals following the administration of iodine. Martin¹³ noted no decrease during its administration in 4 normal individuals, but did observe some decrease in cases of simple goiter, primary anemia and rheumatic fever. Marine¹⁴ has reported a lowering of the metabolism in rabbits as a result of administration of large doses of iodine. This depression of the metabolism to a low level however, appears to be the exception rather than the rule, both in postoperative exophthalmic goiter patients and in normal individuals.

There thus appear to be two definite causes of our cases of temporary low metabolism:

1. A temporary underfunction of the thyroid gland.

2. A temporary reduction to a level of metabolism which is normal for the individual, or else is due to some unknown abnormality other than myxedema, the

subsequent rise in metabolism being due to mild thyrotoxicosis.

In our experience, whenever a depression of the metabolism to a level 15 to 20 per cent below the average normal is due to an underfunction of the thyroid, typical signs and symptoms of myxedema almost invariably become evident if the low metabolism lasts two to three months. We, therefore, are reasonably certain that some of the metabolic depressions caused by iodine were not due to an underproduction of the thyroid hormone. We do feel, however, that several of the temporary depressions, whether produced by iodine or not, may have been due to an underfunction of the thyroid, which, because of its short duration, was not detectable clinically. This assumption is based on the slowness with which myxedema often recurs when thyroid therapy is omitted. The effect on metabolic rate is often well marked before any manifestations of myxedema are evident on physical examination.

This explanation probably applies to all cases in which the lowest metabolism recorded was below minus 25 per cent. It probably also applies to several in which it was above minus 25 per cent. The number of cases due to each cause can be definitely determined only by following the patients until their metabolism has reached a stationary level.

If our explanations for temporary low metabolism be correct, it follows that several of the cases may eventually have a permanent low metabolism without myxedema, with the intervening period of higher metabolism representing a thyrotoxic level. The patient whose data are charted in Figure 5 thus may be regarded as a connecting link between these two types of low metabolism.

If patients whose metabolism is normally low can develop exophthalmic goiter, it follows that thyrotoxicosis may exist with a standard normal metabolism. Thus, in order to judge the true elevation of the metabolism in patients with thyrotoxicosis, it is necessary to know their normal metabolic level.

SUMMARY

Sixty-six cases have been collected showing a basal metabolic rate below minus

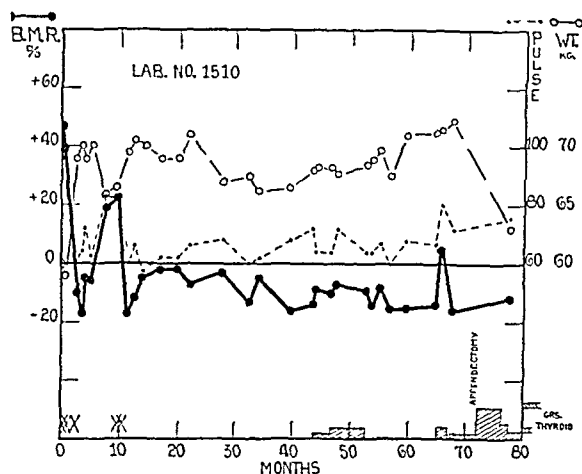


FIG. 5. Miss L. B., aged twenty-three years. Temporary low basal metabolism without myxedema followed by permanent low basal metabolism without myxedema, occurring after roentgen-ray treatment (X) for exophthalmic goiter.

15 per cent after treatment for toxic goiter. Only 11 of these had signs and symptoms that were characteristic of definite myxedema. In 3 cases the myxedema was temporary and in 8 presumably permanent.

It is considered significant that in 1 of the temporary cases the myxedema was shown to be the result of the postoperative administration of iodine.

Of the remaining 55 non-myxedematous patients, in 26 the low metabolism was temporary and in 21 permanent; in 8 its type could not be determined.

It is shown that the temporary depressions of the metabolism, unassociated with myxedema, were probably due to two things:

1. A temporary thyroid deficiency, which, because of its short duration, was not detectable clinically.
2. A transient return to the normal metabolic level of the individual.

Permanent low metabolism without myxedema following thyrotoxicosis appeared in most instances to represent a return to a normal metabolic level, which was probably low even before the development of the disease.

The interpretation of the degree of

basal metabolic elevation in thyrotoxicosis is, therefore, directly affected by the level of the patient's normal metabolism.

In most of the patients with myxedema

the basal metabolic rate was below minus 25 per cent. In all the patients whose low metabolism could be regarded as normal, the rate was above minus 25 per cent.

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ANESTHESIA FOR THYROIDECTOMY IN CHILDREN*

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THE problems connected with the administration of anesthesia to children with severe hyperthyroidism concern not only the persons actively engaged in the operation, but also the whole hospital staff; for these problems are related not only to the time of operation but must be considered from the time the patient first enters the hospital. Thus, from the standpoint of the anesthesia as well as in other respects, the general management of the case is of the utmost importance.

Hyperthyroidism is not common in children under fourteen years of age, but when it does occur it often assumes a very severe form. It is characterized by extreme nervousness, alarming tachycardia and a marked tendency toward the development of acidosis, all of these symptoms being much more pronounced than in adult cases in which the disease is apparently of the same degree of severity. Consequently these cases are more difficult to handle and have a higher operative mortality rate.

These patients enter the hospital in a very apprehensive state of mind, a condition which is partly due to the disease, but is often in great part due to a fear of operation which has been instilled into them while other forms of treatment have been carried out, I recall one little boy who was terrified if more than one person was in his room at a time, because he feared that someone whom he could not watch would produce a knife from his pocket and would operate. This state of mind is often brought about by misguided and oversolicitous parents, who present as great a problem as is presented by the patient himself.

The general management of these patients should be as simple as possible.

Unnecessary procedures such as basal metabolic tests should be omitted, as they only disturb the patient. The attendants should be selected for their cheerfulness, tact and interest in children, and they should be as few in number as possible, and if possible, they should not be changed.

The first essential is to gain the patient's confidence and to make him realize that he will suffer no pain. Here begins the rôle of the anesthetist. She should be a person of friendly disposition, possessed of cheerfulness, firmness and tact, and she should be very proficient in the administration of nitrous oxid anesthesia. She should visit the patient frequently and become his staunch friend. Bringing toys to the child and reading aloud to him are very effective means of gaining his confidence, and of instilling a feeling of safety in her presence.

Some incentive must be found that will arouse a desire in the patient to get well. Thus for the boy the suggestion that he should become able to return to school and engage in athletics generally answers this purpose. The next step is to make the child realize that though everything possible is being done for him, it is necessary for him to help in his own treatment. He will soon take pride in his progress; in the realization that things which frightened him badly on admission have scarcely any effect, that his pulse does not become as rapid as formerly during treatments, and that he sleeps well. Once this stage has been reached, a great deal has been accomplished.

These children are often precocious and will ask about the operation. It is better not to discuss it at length. In the case of some patients it is well to explain that an operation is the only means of getting well, taking care to emphasize the

* From the Cleveland Clinic. Submitted for publication November 5, 1928.

fact that they will feel no pain. With others, to whom "operation" is a terrifying word, it is better to use such terms as "treatment," "injection," "ligation of blood vessels" etc. It also helps greatly to show such a child a younger patient who has recently had the operation, for this often stirs in them a desire to make even greater progress.

As soon as the patient has become fairly quiet the anesthetist should accustom him to the gas machine and to the operative position. Daily inhalations of oxygen should be given, and they may be referred to as "treatments to quiet the nerves" or "to make the operation easier." At first the patient should be allowed to see the machine at a distance; then, as his fear is dispelled and his interest is aroused, it may be brought closer. In a few days the mask may be applied to his face. Tying the hands to the side is often a difficult step; however, it can invariably be accomplished if it is approached with tact, ingenuity, and some degree of firmness. Finally, the eyes should be covered and the neck cleaned and draped. The importance of maintaining mental contact with the patient during these procedures by continuous conversation cannot be overemphasized.

In respect to the anesthetic agent itself, ether is absolutely contraindicated because of its tendency to precipitate an already impending acidosis. Nitrous oxide-oxygen is the most satisfactory agent at our disposal; however, even this must be used sparingly, as it has the same tendency to cause acidosis, though to a much less degree. We have found the safest method is to induce a state of analgesia with an inhalation of nitrous oxide-oxygen and to use local infiltration of a $\frac{3}{4}$ per cent solution of novocaine.

The plan of action for the operation itself is, in brief, as follows: During one of the daily inhalations the operating team

quietly enters the patient's room, unknown to the patient. The operative field is prepared and draped, just as it has been done on preceding days. A light anesthesia is then induced by means of the gas, and a complete novocaine block is gently but rapidly made, both the operator and the first assistant inserting the needles. The patient is then gradually brought to the stage of analgesia and the anesthetist regains mental contact with him. The operation proceeds without disturbing the patient. Should it be necessary to cause pain by traction or otherwise, the anesthetist is warned so that she can momentarily induce a light anesthesia. Ligatures for hemostasis can always be placed and the wound can always be closed under analgesia. The state of analgesia is continued until the operating team has left the room, the hypodermoclysis needles have been inserted and a hypodermic injection of morphine or of codeine has been given. The room is then darkened and only one attendant remains.

By such a procedure the patient is almost completely protected from psychic trauma and physical pain without the deleterious effects of a long (general) inhalation anesthetic. The great danger of severe acidosis is reduced to a minimum, certainly as far as the anesthetic is concerned, and the convalescence is accordingly much smoother. The patient is always greatly relieved to know that the operation is over and that he can go home in less than a week.

The same plan is followed whether ligation, lobectomy or thyroidectomy is done. After the patient has once been satisfactorily prepared for the first operative procedure, it is much easier to prepare him for a second or a third. It is obvious that a great deal of responsibility is placed upon the anesthetist, and should she not perform her part satisfactorily the plan will result in a dismal failure.



SPINAL ANESTHESIA—VOLUME CONTROL TECHNIQUE*

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MUCH interest has been aroused by recent developments in spinal anesthesia, but there still exists considerable skepticism on the part of many surgeons as to its reliability and safety.

A technique has been developed at the Jackson Clinic which makes possible a definite control of the height of anesthesia on the body without the introduction of any substance other than novocaine into the spinal canal. This technique also prevents the wide variations in blood pressure and the nausea, vomiting and related discomforts which frequently occur with most of the methods now in general use.

Corning, a neurologist, first attempted to relieve the pain of a diseased spine in 1885 by injection of a solution of cocaine. Quincke, in 1891, was the first to withdraw spinal fluid by lumbar puncture; Bier in 1899 successfully injected cocaine into the spinal canal, and Tait and Cagleri in 1899 used intradural injections of cocaine for surgical anesthesia; the method enjoyed a brief popularity but was soon abandoned because of frequent disastrous results. With the discovery of the less toxic drugs, stovaine by Fournau in 1904 and novocaine by Einhorn in 1905, spinal anesthesia was again attempted by Braun and others with more favorable results.

In America Babcock and Labat have been for years the foremost exponents of the method and have reported several thousand cases, with an extremely low morbidity and mortality. The majority of spinal anesthetics until recently have been administered in a few large surgical centers. Surgeons elsewhere, not fully realizing its numerous advantages, were not familiar with the method. Pitkin has recently

popularized spinal anesthesia through the routine prophylactic use of ephedrine, which adds greatly to the safety of the method, together with gravitation of light and heavy solutions of novocaine first suggested by Barker in 1907 to control the extent of anesthesia.

Many of the recorded accidents with spinal anesthesia have been due to an inadequate understanding of the hydrodynamics of the spinal fluid and of the technical procedures involved. Lack of preparation to meet the emergencies that may arise have added an occasional fatality. The majority of deaths have been among patients moribund at the time of operation, and undoubtedly would have occurred with any form of anesthesia. Against these may be counted many patients who have survived major surgery under spinal anesthesia, for whom any other form of anesthesia probably would have been fatal.

PHYSIOLOGY

Novocaine in solution, when introduced into the spinal canal, is rapidly absorbed by nerve tissue and blocks the passage of sensory or motor stimuli. When it is diffused upward in the dural canal to a level necessary for the performance of abdominal surgery, a large proportion of the spinal nerves that supply motor stimulation to the sympathetics by way of the white rami communicantes are paralyzed, causing vasodilation and a fall in blood pressure. The extent of this fall in blood pressure has been found to hold a direct proportion to the extent of sympathetic paralysis, which in turn may be gauged by the height of anesthesia on the body.

The Trendelenburg position was first used to combat this fall in blood pressure and is still being used routinely by Bab-

* From the Division of Surgery, Jackson Clinic. Submitted for publication May 7, 1929.

cock and Labat without other stimulation. This causes blood to gravitate to the brain and prevents cerebral anemia in spite

off and the sympathetics regain control. Ephedrine admirably fulfills the foregoing requirements. Its use in spinal anesthesia

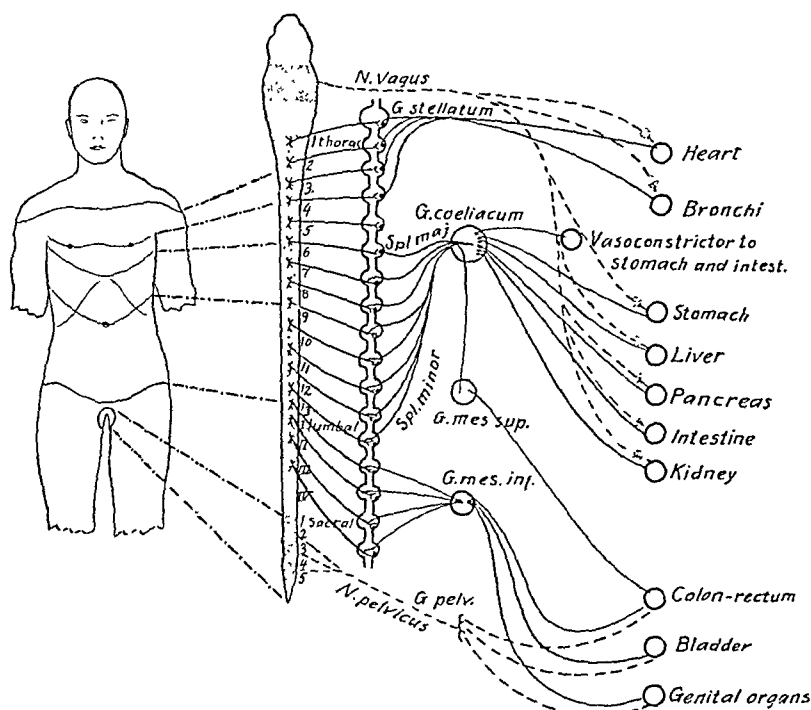


FIG. 1. Diagram showing extent of sympathetic paralysis compared with level of anesthesia on body. Sympathetics are vasoconstrictors, while parasympathetics (broken lines) are vasodilators. Anesthesia to clavicle involves paralysis of all sympathetics and maximum fall in blood pressure which may be prevented by maximum prophylactic dose of ephedrine. Anesthesia to umbilicus involves paralysis of about half of sympathetics, therefore half maximum dose of ephedrine is given. Perineal anesthesia does not cause sympathetic paralysis, hence no ephedrine is given.

of the general vascular relaxation, but allows stagnation in the dependent portions of the body and anemia in the uppermost.

The Trendelenburg position in most instances will suffice to save the patient from death by cerebral anemia, but the occasional case of severe myocardial damage allows stagnation to such an extent that emergency cardiovascular stimulation may become necessary. Adrenalin has been used for this purpose, but its action is so fleeting that repeated injections are necessary and the patient's vascular instability often becomes a source of concern to the anesthetist.

A more desirable procedure is the prophylactic administration of some peripherally acting vascular stimulant which will maintain a normal vascular tone over the entire body until the anesthesia wears

was introduced by Ockerblad and Dillon in 1927. They first gave it orally after the blood pressure had fallen 10 per cent of its original level and later hypodermically. Its prophylactic use before induction of the spinal anesthesia was then tried with such favorable results that it has become the method of choice.

VOLUME CONTROL—THEORY

In a series of 600 cases in the Jackson Clinic, we have found it possible to produce a spinal, or intradural radicular block anesthesia, which can be accurately controlled as to the height of anesthesia on the body. This technique does not involve the introduction of any foreign substance other than novocaine into the spinal canal, and its success depends upon an understanding of the hydrodynamics of

the spinal fluid rather than the effect of gravity upon light or heavy solutions of novocaine. Nothing has been introduced to prevent diffusion of the drug within the spinal canal as it is possible, with an understanding of the physical principles governing this diffusion, to control it accurately enough for practical purposes.

In a review of physical chemistry certain principles governing diffusion of crystalloids in solution will be found, which, when applied to spinal anesthesia, form the basis of a method to control the extent of diffusion of novocaine in the spinal canal, and hence the height of anesthesia on the body.

UPWARD EXTENSION OF ANESTHESIA

1. *The upward extension of anesthesia is directly proportional to the speed of diffusion of the novocaine injected into the spinal canal.*

The diffusion rate of a crystalloid in a preponderantly aqueous solvent is constant when the temperature of the solutions, their proportionate volumes and concentrations, and the amount of mechanical agitation are kept constant.

2. *It is inversely proportional to the rate of fixation of novocaine by nerve tissue.*

Diffusion of a crystalloid in an aqueous solvent proceeds at a definite rate until its concentration is uniform throughout the solvent. Complete diffusion may be prevented by the early removal of the diffusing crystalloid from the solvent. Thus general anesthesia is prevented by the rapid fixation of novocaine from the spinal fluid by nerve tissue, and the extent of diffusion is limited by the rapidity of fixation. The rate of fixation, as observed clinically, is practically constant, therefore, the rate of diffusion must be varied to produce a variation in the extent of diffusion or the resultant level of anesthesia.

3. *It is directly proportional to the volume of fluid injected.*

The larger the volume of fluid injected, all other factors remaining constant, the

higher will the anesthesia extend as a direct result of increased volume displacement and increased mechanical agitation which accelerate the rate of diffusion of the injected novocaine.

4. *It is directly proportional to the speed of injection.*

The more rapid the injection the faster the upward diffusion of the anesthetic agent, due to increased mechanical agitation.

5. *It is inversely proportional to the cerebrospinal fluid pressure.*

The diffusion of any two liquids under pressure is slower than under decreased pressure. When unmeasured quantities of cerebrospinal fluid are allowed to escape, the pressure is reduced an unknown amount and diffusion will proceed to a higher level, proportional to the decrease in intraspinal pressure, and the anesthetist is unable to predict the resultant level of anesthesia.

6. *It is directly proportional to the specific gravity of the solution.*

Increasing the specific gravity of the solution by use of large amounts of novocaine will increase the rate of diffusion by increase of mass and hence inertia of the heavy injected solution. If more novocaine is injected than is required to saturate the nerve tissue to the desired level, diffusion will continue until all the novocaine is fixed or absorbed from the spinal fluid, thus producing a higher level of anesthesia than desired.

7. *It depends upon the position of the patient's body when there exists a difference in specific gravity between the solution injected and the spinal fluid.*

Gravitation of a concentrated solution of novocaine to the tip of the dural canal will produce anesthesia of the perineum only, as will gravitation of concentrated novocaine to the dorsal or cervicodorsal curves produce segmental anesthesia of the upper abdomen and thorax. The Trendelenburg position assumed during or immediately after injection of novocaine will accelerate the rate of upward diffusion while Fowler's position will retard it.

BLOOD PRESSURE CONTROL

As previously mentioned, ephedrine is a peripherally acting vascular stimulant

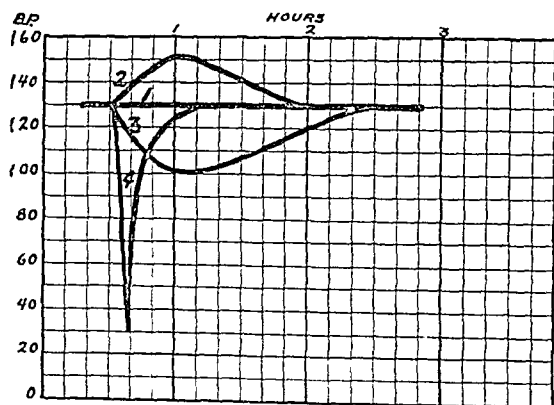


FIG. 2. Typical systolic blood pressure curves. 1. Proper dosage of ephedrine given five minutes before intraspinal novocaine. 2. Moderate overdosage of ephedrine. 3. Moderate underdosage of ephedrine. 4. Ephedrine given after intraspinal novocaine. Sudden vasodilation, nausea, vomiting and faintness. Blood pressure restored to former level by adrenalin and maintained by previously given ephedrine.

which, when properly administered, will maintain a normal vascular tone over the entire body until the anesthesia wears off and the sympathetics regain control.

If the anesthetic agent is diffused to such a level that all the sympathetics are paralyzed, a maximal fall in blood pressure will occur, while if only a portion of the sympathetics are paralyzed, a proportionately less severe fall will occur. In the absence of any method to definitely control the height of anesthesia, when using novocaine crystals dissolved in spinal fluid, the degree of fall in blood pressure to follow could not be predicted. A uniform prophylactic dose of $\frac{3}{4}$ grain of ephedrine is commonly used, supplemented by the Trendelenburg position, adrenalin, or more ephedrine as the occasion seems to demand. This is a definite improvement over previous methods, however, nausea, vomiting and minor variation of blood pressure continue to occur when the anesthesia extends higher than the costal margin.

With the development in the Jackson Clinic of volume control technique and

the certainty of producing anesthesia to any definite level on the body as desired, we were able to predict with a fair degree of accuracy the extent of the fall in blood pressure to be expected. The prophylactic dose of ephedrine was then varied in direct proportion to the degree of vasodilation expected. This has resulted in a much greater vascular stability during anesthesia with less nausea, vomiting and physical discomfort to the patient.

The proper timing of the prophylactic dose of ephedrine has also been found to be an important factor in maintenance of blood pressure as is illustrated by Figure 2. If the spinal anesthesia is induced before absorption of the ephedrine has started, vascular tension is reduced to such an extent that absorption takes place slowly, if at all, and stimulation by adrenalin becomes necessary.

It has been noted that the greatest fall in blood pressure, in spite of the prophylactic dose of ephedrine, occurs in hypertensive cases with sclerosis. The explanation may be offered that while the peripheral vessels may be palpably sclerotic, the splanchnic vessels are rarely as markedly so. As ephedrine is a peripherally acting vasoconstrictor, its stimulation in sclerotic cases is relatively ineffective, and as the less sclerotic splanchnic vessels dilate with onset of anesthesia a great fall in blood pressure is usually experienced. This suggested the use of larger doses of ephedrine or the addition of a small dose of adrenalin. The latter therapy has been used successfully in the few cases met with in this series. Patients with functional or essential hypertension (not encountered in this series) would on a theoretical basis be given a small dose of ephedrine. Hypotensive patients may be given an increased dose of ephedrine which will raise their pressure to a safe level during the anesthesia.

The most important factors in volume control technic of spinal anesthesia are: the aspiration of accurately measured volumes of spinal fluid used as a solvent for graded doses of novocaine, and the

reinjection of the spinal fluid novocaine solution at a constant measured rate without loss of fluid during the manipula-

and the condition of the patient during operation.

Preparation of the patient for operation

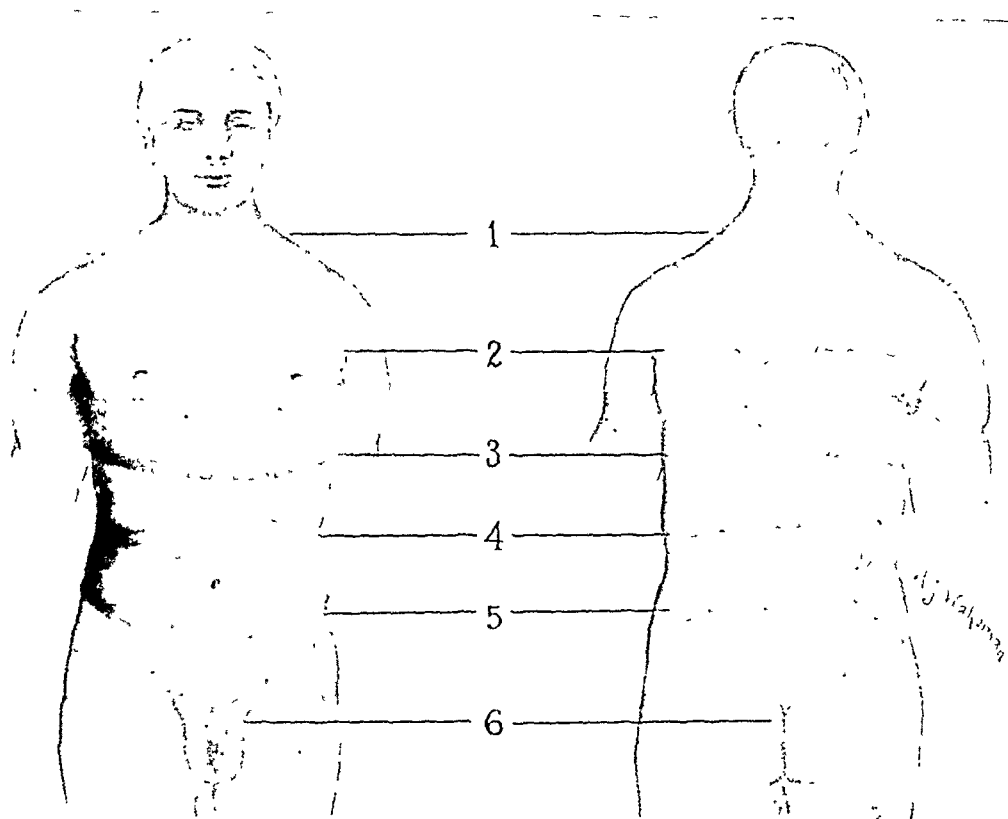


FIG. 3. Various levels of anesthesia. Shaded bands indicate zones of analgesia above anesthesia. (See Table 1 for technique.)

tion. This is facilitated by the use of small caliber (22 gauge) spinal puncture needles which minimize fluid loss and avoid postpuncture leakage and cord trauma. Of equal importance is the properly timed prophylactic administration of ephedrine in amounts directly proportional to the height of anesthesia to be produced, varied also according to the size of the patient and his vascular condition.

A careful record should be kept of all factors varied under control, the extent of the anesthesia produced and its duration, the patient's general condition, and any medication during the operation, with graphic blood pressure and pulse records. These records must be studied and compared to obtain a full realization of the effect of variation of any given factor upon the resultant level of anesthesia

under spinal anesthesia includes at least twenty-four hours of rest in bed, adequate hydration, and the usual laboratory and physical examinations. The following standardized routine orders are carried out in the preparation of all patients for major operations:

1. Fluids should be forced to at least 3500 c.c. during twenty-four hours preceding operation.
2. Thorough cleansing enema on evening before operation.
3. Ten grains of sodium barbital by mouth one and one-half hours preceding operation.
4. Pantopon, grain $\frac{1}{3}$, and scopolamine, grain $\frac{1}{150}$, by hypodermic one hour before operation.
5. Stop up patient's ears and cover eyes, keep room quiet.

The technique shown in Table I will produce anesthesia to the indicated levels in the average adult weighing 150 pounds.



FIG. 4. Anesthesia of perineum (shaded area) as induced for obstetrics, perineal and rectal surgery. (See Table I for technique.)

TABLE I
TECHNIQUE

Level of Anesthesia (See Fig. 3)	Volume of Novocaine Spinal Fluid Solution Cc	Average Novocaine Dosage in Mg.	Average Length of Anesthesia in Hours	Injection Time in Seconds	Ephedrine Dosage in Mg.
1	3.5	300	1½	17	95
2	3.0	200	1¼	15	95
3	2.5	200	1½	12	80
4	2.0	150	1¼	10	50
5	1.5	150	1½	7	30
6	1.0	100	2	5	0

Volumes, novocaine and ephedrine dosage must be increased proportionately for large individuals and decreased for small ones. Ephedrine dosage is varied from that prescribed in the table in cases of hypotension and hypertension. Any severe fall in blood pressure within five or ten minutes after injection of the novocaine-spinal fluid solution means that an insufficient prophylactic dose of ephedrine was given or that the intraspinal injection was made too soon after the ephedrine was given. If the injection is made five minutes or more after a sufficient dose of ephedrine has been injected *intramuscularly*, this fall will not occur.

TECHNIQUE

1. Record blood pressure on graphic chart.

2. Prepare surgical field, cover with sterile towel, strap towel down with adhesive straps.

3. With the patient in the right lateral position, back bowed out, knees up, neck flexed, and steadied by an assistant, the back and upper buttock is thoroughly scrubbed with ether and alcohol, dried and painted with 3 per cent tincture of iodine.

4. Inject ephedrine *intramuscularly* in buttock noting time of injection. (For dosage see Table I.) Massage site of injection thoroughly. Ephedrine is not given when perineal anesthesia only is desired.

5. After palpation of a convenient lumbar interspace (usually the second or third) produce local ischemia by firm pressure of the thumbnail for ten to twenty seconds. Then quickly introduce a 22-gauge spinal puncture needle through blanched area which has been rendered temporarily anesthetic by pressure. Continue introduction of needle slowly through interspinous ligament into dural sac, which is recognized by a slight snap and subsequent lack of resistance. Novocaine skin infiltration may be used if so desired.

6. Remove stylet of spinal needle. When fluid is seen welling up into hub of needle, quickly attach syringe without loss of fluid and slowly aspirate the required volume of fluid. (See Table I for volume.) Detach syringe and quickly replace stylet.

7. Attach another needle to syringe and transfer spinal fluid to ampule of novocaine crystals (For dosage see Table I). Dissolve crystals by barbotage, aspirate solution into syringe, detach needle and express any air bubbles.

8. Five minutes after ephedrine was injected, remove stylet from spinal needle, quickly attach syringe and inject solution at a rate of 1 c.c. in five seconds without barbotage.

9. Spinal needle is then withdrawn, still attached to syringe, and with a sterile

sponge held over the site of puncture, the patient is placed in position for operation.

10. Blood pressure readings are taken

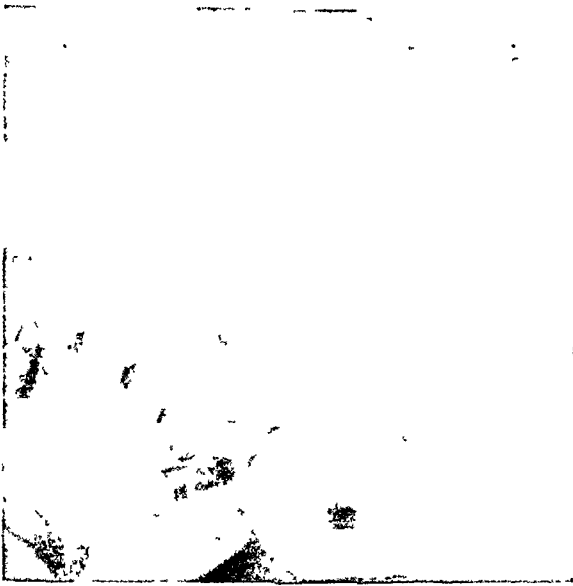


FIG. 5. Accurately timed intraspinal novocaine injection. (Note wrist watch.)

and recorded while draping the operative field, and the patient is told that he may expect a sensation of numbness to follow shortly.

11. The level of anesthesia is determined by pin-prick five minutes after

of the novocaine in the nerve tissue by that time. The level of anesthesia may be raised if the Trendelenburg position is taken sooner. If too large a dose of novocaine has been used, complete absorption takes a longer time and Trendelenburg position in ten minutes may allow gravitation of the unabsorbed remainder, thus raising the level of anesthesia on the trunk.

A competent anesthetist should be with the patient throughout the operation and attend to his mental and physical comfort. Blood pressure and pulse rate are recorded at frequent intervals. Pantopon or morphine may be administered during the operation if the preoperative sedation is deemed insufficient.

Ephedrine and adrenalin in ampules should be kept ready for hypodermic injection in case any considerable fall in blood pressure should occur.

Ephedrine sulphate in ampules containing $\frac{3}{4}$ grain in 1 c.c. solution (Lilly) and novocaine crystals in ampules containing 100 or 200 mg. (Metz) have been used throughout this series of cases.

Luer-Lok 2 c.c. and 3 c.c. syringes and B-D Erusto 22 gauge spinal puncture needles with 45° bevel point have been

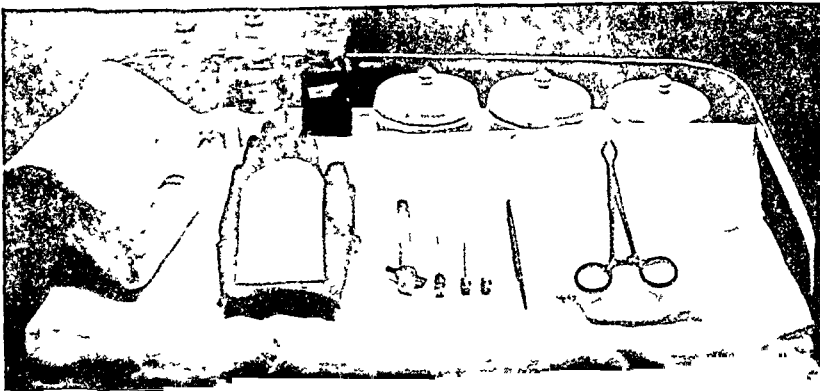


FIG. 6. Sterile outfit, opened and ready for use. Novocaine crystals and ephedrine in ampules are kept in alcohol in cups on table. At left is unopened outfit as it comes from autoclave.

injection and recorded. Operation is then allowed to proceed. After ten minutes the level of the table may be changed (Fowler's or Trendelenburg) to suit the convenience of the surgeon. No change in level of anesthesia results, which indicates fixation

found most satisfactory. They may be cleaned by rinsing in distilled water, wrapped in a towel with gauze sponges, gloves and powder, a file and a lifter for the ampules, and autoclaved for twenty minutes at 20 pounds pressure. Several

such sterile outfits are always kept on hand. Ampules of novocaine crystals, ephedrine and adrenalin are kept in small covered cups immersed in 70 per cent alcohol until used.

INDICATIONS AND CONTRAINDICATIONS FOR SPINAL ANESTHESIA

Spinal anesthesia, which has long been recognized as the safest anesthesia for use in cases with severe pulmonary, cardiac, renal or hepatic complications, is certainly the safest anesthesia to use in the average case. The extent to which spinal anesthesia can be utilized in surgery is limited only by the anesthetist's skill and experience, and probably in the near future will almost completely replace inhalation anesthesia.

Hypotension, which in the past was a formal contraindication to spinal anesthesia, may now be successfully overcome by the judicious use of ephedrine. Dehydration, which is a contraindication to surgery, may be effectually overcome by the subcutaneous or intravenous administration of fluid.

COMPLICATIONS OF SPINAL ANESTHESIA

The most common immediate complication of spinal anesthesia is a slight to moderate fall in blood pressure, usually accompanied by nausea and occasionally vomiting when systolic pressure falls below 85 mm.

When due to an insufficient prophylactic dose of ephedrine, nausea may be relieved by the injection of 2 or 3 minims of adrenalin which quickly restores the blood pressure to its former level. Nausea, caused by too vigorous mesenteric traction, is usually accompanied by a slight fall in blood pressure which quickly regains its former level with cessation of the nausea when the traction is stopped. Stimulation by adrenalin in this case will raise the blood pressure but will not relieve the nausea. Adequate incision and gentle handling obviate this difficulty. Psychic nausea is unaccompanied by a fall in blood pressure and may be prevented by avoiding the

clatter of instruments, loud conversation, and noises which might disturb a conscious patient. Even a nervous patient may be comfortably carried through a major operation if sufficient preoperative sedation has been given, or if the anesthetist will keep the patient's mind off the operation by means of an interesting conversation.

Convulsions are reported in the literature to have followed injection of novocaine into the venous plexuses around the cord. Careful lumbar puncture and the administration of 10 grains of sodium barbital by mouth one and one half hours preoperatively will prevent this occurrence.

Respiratory and cardiac failure have been reported with the higher anesthetics, but have not been encountered in this series. Of great interest in this connection is the work of Jonnesco and of Koster who have produced general surgical anesthesia, by the intradural injection of novocaine and neocaine, permitting operations on the head, neck and thorax, without the occurrence of respiratory or cardiac failure. This may be due to gravity keeping the relatively heavy solution of novocaine confined to the posterior position of the spinal canal as it diffuses cephalad, thus anesthetizing the sensory roots only at the higher levels. This may be duplicated by the injection of colored novocaine solution into a saline-filled horizontal glass tube.

Postanesthetic headaches may occur, due to withdrawal or loss of large amounts of spinal fluid, either at the time of puncture or later through a gaping puncture wound made by a large needle. Needles of 22 gauge with a short bevel obviate this difficulty. When perineal or intradural caudal anesthesia has been induced for some minor procedure, such as cervical cauterization, circumcision in the adult, or cystoscopy, headaches, due to leakage of spinal fluid through a small but unhealed puncture in the dura, have followed when the patient was immediately allowed up and about. These headaches have been avoided by requiring bed rest for at least twelve hours.

Meningismus, which is the result of the injection of a sterile irritant into the spinal canal, may follow the use of imperfectly cleansed syringes or needles.

Meningitis is due to contamination of solutions, syringes or needles.

Transient paresthesias are reported to occur, usually being limited to the perineum or legs, and are usually caused by trauma to the nerve roots by a large calibre long beveled needle.

FAILURES TO OBTAIN ANESTHESIA

Most authorities on spinal anesthesia, particularly De Takats, admit a failure to obtain anesthesia in 5 or 6 per cent of their cases. Two explanations of failure may be offered. First, that the injection of novocaine was extradural, second, idiosyncrasy to novocaine. The first explanation seems to be the most likely as in 4 cases in this series, after complete failure to produce anesthesia, a second injection was made by the same operator and a satisfactory anesthesia was obtained. These failures were undoubtedly due to slight movement of the needle after successful puncture and withdrawal of the fluid, the tip of the needle lying partially or wholly outside the dural canal during injection.

BENEFITS OF SPINAL ANESTHESIA

Relaxation of the abdominal wall, which is more complete than that obtained under any other anesthesia, is appreciated by the surgeon in any case, but especially so in cases of ruptured viscera, such as a ruptured gangrenous appendix or perforated gastric or duodenal ulcer. The necessary surgical procedure may be carried out, without disseminating infective material throughout the surrounding peritoneum, by fighting the labored respiratory efforts of an "ether patient" and forcibly packing away distended intestines.

The intestines under spinal anesthesia are contracted, owing to preponderance of vagus or parasympathetic tone in

the absence of sympathetic inhibitory stimuli. The abdominal walls may be retracted upward with only slight traction,



FIG. 7. Appendectomy in six-year-old patient.

and the entire abdominal cavity may be inspected before any manipulation. This condition of "abdominal silence" is a revelation to the surgeon not accustomed to spinal anesthesia, and has been termed a "surgical paradise" by a recent convert to the method.

Spinal anesthesia constitutes an effective block to pain originating in the operative field and hence removes this possible causative factor in the production of shock. Surgical shock has not occurred in this series of cases.

There is little or no postoperative nausea, vomiting or distention, therefore, less strain on the suture line. This results in less pain in the region of the incision and less chance of occurrence of postoperative hernia.

Paralytic ileus has been overcome by the use of spinal anesthesia to interrupt the reflex sympathetic inhibition of peristalsis which characterizes this condition.

Postoperative respiratory complications occur one seventh as frequently as after inhalation anesthesia.

No demonstrable damage to the liver or kidney has ever followed spinal anesthesia.

The patient is able to take fluids and food before, during and immediately after an operation. Catheterization has thus been much less frequent in this series of cases than after ether anesthesia.

The burdens of the nursing staff are lightened considerably as patients after

operation are returned to their rooms conscious, cheerful and coöperative.

The appreciation of the patient, who has previously had an ether anesthesia, demonstrates that from his viewpoint the method is commendable.

CONCLUSIONS

1. Spinal anesthesia, when skillfully administered, provides complete absence of pain, complete relaxation, and facilitates the rapidity and ease with which any surgical operation may be performed.

2. Convalescence after surgery performed under spinal anesthesia is attended by far fewer complications and enjoys a definitely lower mortality rate than after inhalation anesthesia.

3. Spinal anesthesia is rapidly growing in favor and in time will be used more than inhalation anesthesia.

4. Volume control technique for induction of spinal anesthesia has been used in 600 cases, and has proved unusually satisfactory and reliable. With this technique we are able to produce anesthesia to any desired level on the body without resorting to the introduction of any substance other than novocaine into the spinal canal.

5. The properly timed prophylactic administration of ephedrine, in amounts directly proportional to the height of anesthesia to be produced, will maintain a vascular tone during anesthesia which closely approximates the patient's normal.

I wish to thank Dr. R. H. Jackson and his associates of the Jackson Clinic for their coöperation and encouragement in the application of this technique to general surgery.

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TORSION OF NORMAL FALLOPIAN TUBE*

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THE first published report of torsion of a tube is credited to Bland-Sutton⁹ in 1890. In the following year Pierre Delbet¹⁶ cited a case. Subsequently, cases were reported by Taylor⁵⁵ and Bell⁷ in England, Russel,¹² Hurst²⁶ and Storer⁵² in the United States; Straganoff⁵³ and Warneck⁵⁸ in Russia; Veit,⁵⁷ Fritsch,¹⁸ Sanger,⁴⁵ and Arthur³ in Germany; Hartman and Reymond,²¹ Leguen and Chabry,³² Pozzi³⁹ and Lejars³³ in France. In 1899 Praeger¹⁰ reported his study of collected cases of tubal torsion and in 1900 Cathelin¹¹ tabulated 41 cases which he found in the literature. The latter made a very exhaustive study of the etiology and pathology of this condition. A critical survey of the original references of these tabulated cases reveals the inclusion amongst them of some reports which do not rightly belong to a review of tubal torsion. For example, he included a case of tubal necrosis with fatal rupture which occurred during typhoid, no mention being made of torsion.

Most of these cases are torsion of a hydrosalpinx or of an ovarian or tubal tumor. Anspach² in 1912 reported 95 cases of torsion of Fallopian tubes in conjunction with ovarian cysts with twisted pedicles, hydrosalpinx, tubal pregnancy and neoplasm. He included 31 of Cathelin's cases.

Spontaneous, non-inflammatory torsion of the Fallopian tube is apparently of rare occurrence. Excluding torsion secondary to pathologic lesions of the tube or ovary or both, and excluding torsion involving the ovary with the tube, only 11 authentic cases of primary torsion of supposedly normal tubes in nulliparous women have been found in the literature. Casagrande,¹⁰ Allen,¹ Eunike,¹⁷ Laemmle,³¹ McIlray,³⁴ Michael,³⁵ Michel,³⁶ and

Schwarzwall⁴⁷ report cases in patients who have borne children, but in these instances because of the pregnancy there is a greater possibility of some primary pathological lesion of either tube or ovary being the underlying basis for the torsion. Davies,¹⁵ Auvray,⁴ Schweitzer,⁴⁸ Rogers,⁴¹ Hausen,²² Stark,⁵¹ Darner,¹¹ Jefferson,²⁷ Gillies,²⁰ Gabe,¹⁹ and Heil²³ report cases in nulliparous women. To these cases the one of Hofmann²⁵ should be added because although here the torsion occurred during the eighth month of pregnancy, the very extensive pathological examinations warrant the classification of this amongst the more carefully selected, apparently normal tubes. The same is also true of Bertone's⁸ case of torsion of a normal tube at the beginning of labor in a full-term pregnancy. The case herein reported is in a virgin sixteen years of age in whom no pathological lesion of the pelvis beyond primary torsion of the terminal portion of the tube could be found.

CASE REPORT

Patient, aged sixteen years, admitted January 19, 1928 complaining of pain in the right lower quadrant with a desire to micturate frequently. Father and mother living and well. No history of febrile diseases. Had measles and whooping-cough in childhood, tonsillectomy at the age of four. Menstruation began at fourteen and one-half years of age, regular every four weeks, flowed six days, dysmenorrhea. The patient had a regular period on October 1, 1927 lasting six days, typical in all respects. The next period on November 7, 1927 lasted two days. On December 7, 1927 there was another period lasting two days. The January period came late on January 13, 1928, lasting six days, during the latter part of which her present illness began.

On the evening of January 17, 1928 the patient experienced sharp pain across her lower abdomen, the pain, sudden in onset, was

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so severe, she could not walk. She neither vomited nor felt nauseated. On the 18th the pain was localized to the right lower quadrant. Cold compresses to the abdomen did not give any relief. On January 19 the pain was still present in the right lower quadrant, and there was a slight elevation in temperature (101°F.). The positive physical findings were tenderness over the right lower quadrant with pressure release tenderness over the entire abdomen referred to the right lower quadrant. It should be mentioned that there were no rigidity, negative Murphy's, negative psoas and negative obturator internus signs. A rectal examination revealed a tender mass in the right lower quadrant apparently hanging from a pedicle which sprang from the uterus. The urinary examination was completely negative and the blood count was: W.B.C. 12,200; polys. 86 per cent; small lymphocytes, 14 per cent. A diagnosis of ovarian cyst with twisted pedicle was made. Laparotomy was performed and the mass in the pelvis was found to be the right tube which, containing some hemorrhagic fluid, was twisted about one and one-half complete turns. The axis of the twist was anemic and showed the torsion spiral described by Kustner. The fimbriated end was free, no adhesions were visible. Distal to the point of torsion, which was at the inner third, the tube was swollen, bluish black in color, and tense. The ovary was not involved in the torsion. There was considerable serosanguinous fluid in the peritoneal cavity. The left adnexa and appendix were normal. Salpingectomy, removing the entire tube down to and including the cornua, was performed and the appendix was also removed. The wound was closed without drainage. Microscopic examination of the tube showed a thick wall in which the fibrous tissue and muscle bundles were separated by multiple hemorrhages. The veins were distended. The mucosa was unrecognizable. A diagnosis of hemorrhagic infarction of the tube was reported. The patient made an uneventful recovery, being discharged with primary union ten days later.

ETIOLOGY

The etiological relationship between ovarian cysts with twisted pedicles, hydrosalpinx, tubal pregnancy and neoplasm, and torsion is easily understandable. In a consideration of the pathogenesis in acute omentovolvulus the author has called

attention to the relationship between chronic inflammation and torsion. While this paper deals with torsion in apparently normal tubes, and while inflammatory lesions in the tubes of virgins are not of frequent occurrence, it must be remembered that a supposedly cured vulvovaginitis may flare up in adolescence and the tube may become invaded. Systemic infections may be accompanied by local inflammatory lesion of the tubes. Salpingitis has also been reported as a complication of the exanthemata. The absence, therefore, of a definite history of inflammation of the tubes or the state of virginity is not *prima facie* evidence of the non-existence of a chronic salpingitis. Anspach² and Norris³⁷ support this view. It should be remembered that in such cases the opposite tube as well would probably be involved in the inflammatory condition. In 8 of the reported cases the opposite tube was normal, and in 5 of the other 7 cases the condition of the opposite tube was not mentioned. Such infections it seems would seal the fimbriated extremity of the tube, yet in several of the reports the fimbria and ostia were described as normal (Gabe¹⁹). This controverts the claim that spontaneous torsion of the normal tubes never occurs, a primary lesion necessarily being always pre-existent. Since the involved tube is almost always distended with bloody fluid, the contention is that the torsion occurred in a tube the seat of a hematosalpinx or even a hydrosalpinx, the colorless fluid of the latter being colored by a congestion transudate.

Kehl²⁸ reports a case of torsion of the tube in a virgin twenty-one years of age occurring during menstruation, in which tube microscopic examination revealed deposits of calcium which "after ruling out tuberculosis remotely reminded one of oxyuris vermicularis of the intestinal wall." Remembering that the oxyuris has been found in the vaginal vault (Heller²⁴), in the uterus (Simons⁴⁹ and Klee²⁹), in the tubes (Tschamer⁵⁶ and Strasser²⁰) and in the cul-de-sac of Douglass in the form of

calcified deposits resembling rice seeds (Chiari,¹² Kolb,³⁰ and Schneider⁴⁶) it is possible that calcific deposits of parasitic origin may produce congestion which in turn leads to hemodynamic torsion. For this reason in cases of torsion of an apparently normal Fallopian tube, it is desirable that careful inquiry be made into the previous history with special reference to symptoms of salpingitis and that a careful pathological examination of the tube be made.

The mechanism by which an apparently normal tube undergoes torsion is very obscure. There are several anatomical and physiological factors which might predispose to twisting of the tube.

The normal tube as it leaves the cornu of the uterus turns downward and backward and then medially toward the uterus. This in itself produces some rotation. It possesses considerable inherent motility by virtue of the contractility of its own musculature. The position of the tube is subject to considerable variation. These movements at times may either diminish or increase the normal angulation. Any increase in the contents of the tube would also tend to produce more angulation.

The dynamics of the neighboring viscera, e.g. the cecum, sigmoid and small intestine, influence considerably the position of the tubes and their angulation. Sanes,⁴⁴ Anspach,² and Smith and Butler⁵⁰ have demonstrated that torsion is encountered three times as frequently in the right adnexa as in the left. This is probably due to the greater amount of room in the right side of the pelvis, since on the left the sigmoid encroaches upon the adnexa, and also the relatively greater peristaltic action of the cecum and ileum as compared with the sigmoid.

The rhythmic movements of the tubes described by Corner¹³ have been substantiated and are now easily demonstrable by lipiodol injections. Exaggeration of these movements might easily produce changes in the venous system of the mesosalpinx similar to those necessary to conform with

the following mechanism of torsion. That such exaggeration is possible is evident from the observations of violent contractions of the tubes which are visible at laparotomy under spinal anesthesia.

The mesosalpinx acts as a pedicle for the tube. In this pedicle are large veins which are very tortuous and thus considerably longer than the accompanying artery. The artery, inherently, is a more tense structure than the veins and its cord-like structure is relatively increased by the greater pressure of its contained blood. Since the walls of the veins are thin and easily dilatable, the accumulated blood in their tortuosities causes them to assume a more or less spiral course about the unyielding cord, the artery. This results in slight torsion which in turn tends to produce venous stasis, the twisting effect upon the artery being negligible at this stage. The venous stasis lends added blood volume to the already rotating mass, and this favors further torsion. Thus, a vicious cycle is created. The distended tortuous vein mass suspending around the less yielding artery favors twisting and venous stasis; the stasis adds to the size and weight of the twisting mass. Payr,³⁸ in a study of the mechanism of the pedunculated viscera, was able to produce twisting in a model consisting of a wooden disc representing a viscus, suspended by three rubber tubes representing the vascular pedicle. One tube, relatively unyielding and shorter, simulated the artery and the other two, softer, more pliable and longer, simulated the veins. Fluid forced through the three tubes under a uniform pressure caused a rotation of the disc. A similar result was obtained when the spleen was suspended by its pedicle and fluid was forced through the artery and veins under uniform pressure. In further experiments he produced torsion in pedunculated organs in animals by obstructing the venous flow, and he found that the physical conditions in dead bodies corresponded to those prevailing in living organisms, because here, too, he could produce torsion by impeding

venous return when pumping fluid through the vessels. It seems likely that this mechanism is similar to the one producing the acute intra-abdominal type of omentovolvulus not accompanied by adhesions or masses along the border of the omentum, of which Randolph collected 23 instances.

The presence of a long mesosalpinx is in itself an important contributory factor. The longer the pedicle the greater the likelihood of torsion. This is true of all pedunculated structures. No better example is needed than the relation of the length of its mesentery to volvulus of the sigmoid.

Long accessory ostia and hydatids of Morgagni are also factors in the production of torsion. Persistence of the marked tortuosity common in fetal life may contribute to the possibility of the occurrence of torsion. The increased vascularity of the pelvic organs during menstruation, especially congestion in the large worm-like masses of veins frequently found in the mesosalpinx, also probably predisposes to torsion. Such congestion may help explain the relative frequency of occurrence of torsion of the tube or the twisting of the pedicle of an ovarian cyst during menstruation, pregnancy, or the puerperium.

Sampson in his study of endometriosis has presented considerable evidence supporting the view that there frequently is a reflux of menstrual flow through the tubes. Retention of some of that blood in the distal third of the tube might produce a bulbous swelling. This swelling might well be the starting point of a tubal torsion, similar to the swelling which, occurring at the free border of the omentum as a result of inflammation, is responsible for omentovolvulus.

Any of the factors which influence the position or the motility of the tubes or any combination of them may produce venous stasis in the mesosalpinx. It is then that there is created a condition favorable for the Payr mechanism of torsion. At present this is probably the best explanation for the occurrence. Yet, the best indication of the shortcoming of any or all explanations,

and the complexity of the factors which are held to be causally responsible, is the frequency with which each of the supposed etiological factors occurs in comparison to the great rarity of torsion.

PATHOLOGY

The portion of the tube affected by the torsion appears as a swollen, edematous, purplish-red, pear-shaped tumor. At the heavier end the fimbriated extremity with the fimbriae spread out star-fashion may be recognized. At the other end of the tumor can be seen the twist which in 44 cases of tubal torsion from various causes studied by Storer⁵² showed from one to five and one-half turns, the average being one and eight-tenths. The number of turns does not appear to have any great influence on strangulation, which depends more on the length and size of the pedicle. The degree of strangulation determines the color of the mass.

The wall is thickened beyond what is expected since the lumen contains fluid which would, in a hydrosalpinx, cause thinning of the wall. The fluid is hemorrhagic in character.

Microscopically, the mucosa is thinned out so as to be unrecognizable. The veins are markedly distended, and there is a general extravasation of blood into the tissues widely separating connective tissue and muscle fibers. The bordering mesosalpinx is also involved in the strangulated appearance and microscopically it shows numerous blood-filled spaces interspersed between strands of connective tissue.

SYMPTOMATOLOGY

The symptoms of this condition are very similar to those of an ovarian cyst with a twisted pedicle. In support of the idea that torsion may be produced by forces which will change the position of the tube, it has been noted that frequently the symptoms are precipitated by some sudden muscular effort changing intra-abdominal pressure, such as lifting, stooping, defecating, urinating, etc. Complete acute torsion begins

with a sudden, sharp and intense pain in the involved iliac fossa. The onset may be so sudden as to cause complete collapse. In these cases it is found that the total vascular occlusion is immediate. From this extreme, the character of the pain is subject to all gradations down to a steady dull ache with exacerbations. The initial pain may be epigastric, midabdominal or pelvic with final localization over the site of torsion. If the rotation is slight, the pain may be very mild, and if the rotation is intermittent (because it is incomplete and readjustment occurs), the pain is intermittent and the history of such pain may be extended over a long period of time.

The pain is accompanied by nausea and vomiting in the large majority of cases. It seems as though the shock of the torsion diminishes the excitability of the splanchnic nerves. This splanchnic inhibition allows the parasympathetic impulses full sway and violent peristaltic movements ensue.

As soon as complete torsion occurs sero-sanguinous fluid develops in the peritoneal cavity. Peritonitis sets in as a result of chemical irritation. Usually distention and constipation follow. The intestinal wall loses tone and paralytic ileus is established. As the condition progresses gangrene develops. If infection of the gangrenous mass occurs, the signs and symptoms of peritonitis soon overshadow the previously painted picture.

Urinary disturbances are quite common. These are reflex and may be frequency, dysuria, or anuria due to bladder retention. They do not however predominate the picture.

As has previously been mentioned there is apparently a direct relationship between the menstrual function and the onset of torsion due to congestion, etc. With the inauguration of the menstrual function there may be many disturbances. Among these the most common are dysmenorrhea and menorrhagia.

The attack is not ushered in with fever but shortly afterward there is an elevation

of temperature. This is accompanied by a corresponding acceleration of the pulse rate. The violence of the clinical symptoms however is out of all proportion to the small rise in temperature. This is of considerable diagnostic importance being comparable to similar violent cramps and abdominal pain of intestinal obstruction which are also out of all proportion to the rise in temperature and the acceleration of the pulse.

PHYSICAL SIGNS

The appearance of the patient may vary from that of moderate discomfort on the one extreme to severe shock on the other. Tenderness over the lower abdomen, more severe in one iliac fossa than in the other, is a common finding. Soon after the onset of symptoms pressure-release tenderness can be elicited over the entire lower abdomen. Rigidity of the involuntary muscular type is always present over the entire lower abdomen. On pelvic examination a mass of varying size (depending upon the duration of the torsion) can be palpated in the lateral fornix of the affected side. These signs coupled with a moderate elevation of temperature, a correspondingly accelerated pulse rate and a moderate leucocytosis are the only constant physical findings.

DIFFERENTIAL DIAGNOSIS

The diagnosis of torsion of the Fallopian tubes will most frequently be confused with the following conditions: ovarian cyst with twisted pedicle, ruptured corpus luteum cyst, intestinal obstruction, salpingitis, omentovolvulus, appendicitis, pyelitis, biliary or renal colic, and perforation of a viscus. Of these conditions the three which will most nearly simulate torsion of the tube are ovarian cyst with twisted pedicle, omentovolvulus and appendicitis. Ovarian cyst may occur at any age. Smith and Butler⁵⁰ in a recent paper record 25 instances of twisted ovarian tumor before puberty. Darner¹⁴ mentioned an ovarian cyst with twisted pedicle

found in a stillborn child. The diagnosis between torsion of a twisted ovarian cyst can only be made when it is known that there was a cyst of the ovary before the onset of symptoms. Rupture of a corpus luteum cyst of the ovary with intraperitoneal hemorrhage is not an infrequent occurrence in young adults. The symptoms of this condition closely simulate those of torsion of the tube but a pelvic examination revealing the absence of a mass in one of the fornices helps exclude torsion. Omentovolvulus is much more frequently encountered in the fifth decade of life. In the history it is commonly noted that there has been a hernia of the inguinal variety of long standing. The sac may be continually empty, it may intermittently be filled with contents or the hernia may be of the irreducible variety. Careful examination will reveal an elongated pendulous mass in the abdomen. Occasionally it may be determined that the upper portion of this mass spreads out in a fan shape to be lost in the region of the transverse colon. Usually, tympany may be elicited on all sides of the mass. If the strangulating omental mass is in close proximity to the abdominal wall, there is a very definite localized tenderness and rigidity of the abdominal wall.

This condition will more frequently be erroneously diagnosed as appendicitis than any thing else. This is for two reasons: (1) because of the frequency of occurrence of acute appendicitis in adults and adolescents, and (2) because the onset of torsion may be characterized by general abdominal cramps later localizing in one or the other quadrants. This sequence (with localization to the right lower quadrant) is so characteristic of the onset of acute appendicitis, that it has been said to be pathognomonic. A thorough pelvic examination however will decide between the two conditions.

The examination of a catheterized specimen of urine will establish the diagnosis of pyelitis when there is any question between the two conditions. Biliary colic may easily

be differentiated by the negative pelvic findings, by the localization of the pain, by the characteristic accompanying gastrointestinal symptoms during the interval of freedom of attacks and by other clinical examinations such as bilirubin estimation and roentgenographic findings. The pain during the crisis is in the right upper quadrant of the abdomen, is referred to the back and right shoulder frequently, and has a tendency toward paroxysms. Tenderness and rigidity below the right costal margin are prominent features and when the gall bladder reaches such a degree of distention as to be palpable as a mass, it is found to be continuous with the free border of the liver and not easily movable. The inability of the patient to take a full inspiration without pain when the examining fingers are gently pushed up beneath the right costal margin in the region of the ninth and tenth ribs is a constant differentiating sign. Icterus, when present simplifies the diagnosis. Renal colic has a definite natural history, which accompanied by urine, cystoscopic and roentgen-ray examinations is easily differentiated from torsion of the tubes. Intestinal obstruction is easily differentiated by the failure to respond to enemata, by the blood chemistry findings of diminished chlorides, increased CO_2 and nitrogenous elements and also by the response to the therapeutic test of spinal anesthesia or the intravenous administration of hypertonic sodium chloride solution. Salpingitis of specific origin is quite easily differentiated by the history of vaginal discharge, much greater rise in temperature and pulse rate, positive vaginal, vaginal or cervical smears and the absence of a large mass in one of the fornices. Perforation of the stomach or duodenum from ulcer is relatively uncommon in females. Where it does occur usually a history of previous gastrointestinal disturbances can be elicited and the physical signs are limited to the upper right quadrant and epigastrium.

One point cannot be too thoroughly stressed in the differential diagnosis of this

condition, namely, the value of a thoroughly conducted pelvic examination. Perhaps no other method of examination will yield such valuable data in differentiating this condition from all others. Not only will this examination frequently give valuable information pointing to the correct diagnosis, but also it will indicate the proper surgical handling of the patient. The latter is far more important. For example, pelvic findings of a mass in the left adnexa will aid a surgeon considerably by suggesting a midline or left rectus incision rather than McBurney's incision for an erroneously diagnosed acute appendicitis.

COMPLICATIONS

If the condition be neglected, rupture of the tube may occur with the production of a hemoperitoneum and the consequent symptoms of secondary anemia. Should rupture fail to occur and the condition persist, gangrene and necrosis of the tube, followed by secondary infection and peritonitis, will ensue. The tube on the other hand may readjust itself and the acute symptoms rapidly subside. If, however, the readjustment occurs later, there may persist a hematosalpinx which if it becomes adherent to the neighboring viscera may cause gastrointestinal or urinary symptoms. The entire mass may undergo organization and later be the cause of an exploratory laparotomy for the deter-

mination of the origin of distressing pelvic symptoms.

TREATMENT

The treatment of this condition is of course surgical. Because it is unlikely that the correct diagnosis will be made in these conditions, the incision will be either of the right rectus or the midline variety. Where a diagnosis of acute appendicitis has been made erroneously and the torsion exists in the left tube, a McBurney's incision through which exploration is conducted will prove to be a considerable handicap to the operator. Excision of the tube down to and including the cornua of the uterus is the procedure of choice. Rogers records a case of bilateral torsion in which it was possible, because of the short duration of torsion of the left side to preserve the tube. It is advisable, therefore, that the adnexa of the side opposite to the one in which the lesion occurs be thoroughly examined for any abnormalities and if any are found some sort of plastic operation should be done to correct or relieve them. In the ordinary case the abdomen may be closed without any drainage, similarly to the procedure adopted in the removal of an ovarian cyst with a twisted pedicle. In cases of long standing, where gangrene and infection and peritonitis have supervened, drainage may be necessary. This may be either abdominal or vaginal.

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TRAUMATIC SURGERY & THE PROBLEMS OF OLD AGE

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THAT an aged person is a poor physical risk is so axiomatic that no contrary argument is needed, and in no field of medicine or surgery is this truism more applicable than in traumatic surgery.

An old person sustaining an injury apparently superficial or trivial becomes a subject of concern to the doctor because the element of physical and mental shock is a more important factor than in a middle-aged or young person. Again, the mere matter of placing an aged person in a recumbent position may induce enough pulmonary stasis to cause early fatality. Many of these old people are like a storage battery which each day is able to start the motor and keep recharging by a not too active turning of the wheels. But like an idle battery, they soon run down if the physical and mental stimulus of their daily duty is interrupted.

Of course in using the terms "old" or "aged" we are thinking in terms of years; but we all know patients of sixty-five who have the vigor and push and vitality of patients of forty-five. What then is the aged patient? To me a patient is not as old as his arteries, but rather as old as the neck of his femur; for with old people that seems a vulnerable part of the anatomy. It has been said by our medical colleagues that "pneumonia is the natural end of the aged," and paraphrasing that a surgeon dealing with the aged injured could truly say, "Fractured neck of the femur is the natural end of the aged injured."

If we briefly survey the field of injuries we soon realize that old people, like their juniors, are liable to injury from three main sources, namely, transportation, in-

dustry and ordinary pursuits. In olden times, war was the great injury producer. Later, industry with a host of mechanical devices was the chief factor. Still later the railways became the leading source. But now the automobile is the chief element not only in urban but in suburban communities. The World War made of our profession a group of specialists organized to care for the injured. That experience showed how deficient we were in the care and treatment of trauma, particularly in the management of compound fractures. The Surgeon-General of the Army is stated to have well said that there were hundreds of our surgeons in France who were masters in gastroenterostomy, but only a few score with equal skill in caring for the wounded. There can be no question that in medical progress traumatic surgery has lagged while other types of surgery have progressed. Fortunately there has been an awakening because of the increasing frequency of automobile and other accidents, and now most hospitals are being so staffed that the seniors and not the juniors are in charge of this kind of service. We are on the way to recognize that a broken thigh is just as much an emergency as a broken appendix.

In the aeroplane age just appearing on the horizon the field for traumatic surgery will be still further enlarged, and it does not require a gift of prophecy to predict that the next great advance in surgery will be in the treatment of the injured.

In old people, as in others, wounds, burns, joint trauma and fractures constitute about 75 per cent of the lesions encountered. Before we discuss any of these let us again remind ourselves that in this group of the aged we must focus our atten-

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tion on the individual rather than on the injury; we treat the person rather than the part.

Let us not forget two elements in our approach to the aged injured: one, the physical and psychic shock, and the other, the lack of what Osler so well called "vital rubber." Thus we start our therapeutic journey with a handicap, and if the old model is to carry through, we must manage to drive with skill especially when the going is rough and the traffic is crowded. Not too fast nor too slow; at a leisurely gait. Not shifting gears too roughly, nor needlessly jamming on the brakes. Coasting when we can, and idling along in neutral when the going is bad. Well, say you, that's all right for the old tin Lizzie or Old Dobbin, but it does not apply to an old man or an old woman. There I differ, for after all the human machine responds to the skilled touch, to the firm but gentle hand, to the experienced guide who knows how to avoid the rough places and who is adept in times of physical and mental stress.

Be gentle then with this old model; temper your driving of it to its capacity and humor its special peculiarities which are the outgrowth of years of service.

And now for some familiar precepts in the management of special injuries among the aged.

WOUNDS

The principle of partial or delayed suture is most important. It should be axiomatic that every wound not made with surgical intent is already infected and should be so regarded. If this be true, how important it is to sterilize adequately accidental wounds by the liberal but gentle use of soap and water. We should not rely on antiseptics, however actively they are recommended or appealingly advertised. We know from abundant experience that each decade reaches a furore for some special kind of germ killers. The carbolic of the distant past was succeeded by the bichloride of several decades ago. Then in the decade just passed the chlorines

were in the ascendant here when they had fallen into disuse in France where they originated.

The Dakin's solution of our day was in effect the Labarraque's solution employed by Baron Larrey who was Napoleon's surgeon in the war of 1814. Just now we are in the throes of another era of antiseptics and we are asked to believe that the germs of our generation can be pestered to death by some gay colors. Red seems to be the first on the list, and many a patient comes to us now a medley of an Indian chief and a futuristic nightmare. Some of the profession are seeing red; they are Bolsheviks in their advocacy of this mercurial, forgetting that long ago we abandoned bichloride because it was dangerous as well as useless. This wound rouge, however, has entered the sunset stage also, and in a few years will join the oblivion of many less livid hued predecessors.

If we must use an antiseptic, and for first aid I suppose we must, let us stick to iodine or permanganate which have been standard for years.

Soap and water are the best cleansers and we can safely rely on these when they are available. In the aged it is especially important not to use any strong antiseptics.

Next comes suturing. It is much better to place the sutures and leave them untied until the second or third day, in the interval covering the wound with a sterile dressing. Infection if any, will develop during this waiting period, and if none appears after this lapse of forty-eight to seventy-two hours, we can have the reasonable assurance that all is well.

If we do suture, let it be loosely and let us always drain. To be sure many cases heal kindly without any such bothersome precautions; that is true, but it is also true that many develop a serious cellulitis. In an old person neglect of these precepts, even in a simple scalp wound, may convert an incident into a catastrophe.

BURNS

These injuries exact a fierce toll at the

extremes of age; we all of us dread a severe burn in a child or in an old person. The shock is more profound, the reaction is slower, the reparative properties are lessened. We regard burns as wounds due to thermal, chemical or electrical contact. This means that the treatment is in keeping with wound therapy and hence we do not smear on unsterile remnants or discards like Carron oil or greasy salves. Nor do we apply an impervious dressing of the paraffine type unless the burn is of a minor grade. There are three stages in the life history of a burn, just as there are three stages in the history of any other trauma. First, the stage of onset or the inflammatory stage characterized by redness, swelling, pain. Second, the stage of progression or the secretory stage characterized by exudation of serum or pus or both. Third, the stage of subsidence or the cicatrizing stage characterized by scar tissue formation with or without contractures.

These you say are the stages of any inflammatory process. Yes, they are; and because they have phasal manifestations of different sorts at different stages it is important to modify treatment accordingly. Our plan has been to treat the first stage by wet dressings of sod. bicarb. (5 to 10 per cent). This is continued usually for three to four days. Then a dressing of sterile olive oil and camphorated oil (equal parts) is substituted. At this stage, the exposure of the part to the rays of the sun or to electric light is also practiced. This "exposure" treatment lessens absorption, promotes exudation and decreases temperature. The final stage is often hastened by covering the area with a dressing of scarlet red ointment, 1 dr. to sterile olive oil 1 oz. Continuing the "exposure" method at this stage is good practice.

Now in old persons, shock and pain need prompt attention. We force fluids; we resort to transfusion if needed. Morphine is used often enough to prevent pain, and luminol is used to allay restlessness. At all stages, we mobilize the parts to

avoid postural contractures. We do not favor débridement of the burned area in the aged; and as a matter of fact that procedure has a limited value in patients of any age. The tannic acid method by which wet dressings of this substance are applied is excellent for body burns involving large surfaces. This is virtually a method of chemical débridement because the burned area dries up and exfoliates.

JOINT INJURIES

The penetrating wounds of joints are serious affairs at any age; but in the old patient, a septic arthritis is likely to lead to amputation or death. Osteoarthritis activated by injury may become a very difficult problem especially if it affects the lower spine or the knee. The appraisal of responsibility is another matter of importance. For example, in one case it was obvious that the osteophytes in the roentgenogram are of long standing and that they are evidences of hypertrophic osteoarthritis. The patient, however, said he has never had any pain in his back until he was knocked down by an automobile at this time. How much of his trouble is old, and how much of it is new, and how much of it is chargeable to this trauma? This brings up the whole question of a single trauma, one accident, acting as an aggravating or accelerating element of an old or quiescent ailment. Can a blow on the chest upset the rhythm of an aged cardiac and cause decompensation? Can a minor bruise on the toe of an old diabetic be the cause of the subsequent gangrene and necessary amputation? That patient with established endarteritis of both legs says a recent bump on the shin caused an ulceration that you know may lead to amputation of the thigh. Did it?

Or those cases of malignancy, superficial as well as deep, that are alleged to be solely or partly traumatic in origin. What is to be our attitude as to these?

We approach the solution of these problems by first of all asking ourselves: What usually or ordinarily happens under

the given set of circumstances? In other words, we rely on past experience of our own or that of others. In answer to this self-propounded inquiry we usually find that an injury of the type described has little or no effect on a chronic or latent ailment. If that be true, then we go on to determine whether or not the ailment in question is characterized by periods of accession or remission, and by continued but perhaps unrecognized progress.

In addition we take note of the "time element;" when did this aggravation occur in relation to this injury? From a clinical standpoint is the connecting linkage adequate? Has the progression of the disease been unusual or is the flare of an accepted type?

Finally, if there had been no history of injury, is there anything in your present findings that would lead you to suspect something beyond the ordinary signs of the disease in question?

If now you believe the injury did aggravate, accelerate or activate the ancient disease, you must be prepared to estimate the degree and duration of this advancement, for many of these accident cases bring up medicolegal questions. How much deformity; how much disability? In old people this is a difficult matter to appraise because of their inability at their time of life to seek or obtain other gainful employment in the presence of a physical handicap due to injury. Their period of convalescence is necessarily longer than with a younger person and their recovery is less likely to be as complete.

A decision by a physician entails much careful thought and complete knowledge of all the facts in the case. Our own plan is to allot 60 per cent for *function* perfectly restored, and 20 per cent each for perfect restoration of *union* and for *contour*. By *function* we mean ability to perform; by *union* we mean the state of repair; by *contour* we mean the external appearance. In other words we judge our end-result in measurable terms based on the two

essentials of *actions and looks*. Cattle or dogs are judged by the same standard of arbitrary values, and I submit that we should be able as physicians to agree on some set of standards so that our opinions will cease to be individual guesswork. How much better to agree in terms of figures than in terms of adjectives or adverbs! You call the result good; I might call it fair, for our standards are not the same and never can be unless we previously adopt the same elements as determinators.

FRACTURES

Naturally fractures of the lower extremity arrest our attention first; and of these fractured neck of the femur claims first place.

When an old person has a broken bone there are certain important steps in treatment, and these may in part be listed as follows:

(1) *General Physique*. Is this particular patient capable of enduring the usual regimen for the injury he has sustained? Can any anesthetic be administered? Is this a case for mobilization or immobilization? Can this patient remain recumbent? Is there any contraindication to skeletal traction, or to open reduction? Do we here and now resolve to treat the individual and relatively disregard the injury? Let us make no mistake at the outset by rating our patient by the calendar of years rather than by the reaction of tissues. My patient of a year ago with a fractured neck of the femur was by the calendar nearly ninety-two years old; but she was up and around in a walking caliper fourteen weeks after her fall.

(2) *Type of Fracture*. Is it a Type I? That is, do the fragments overlap? If so, then our plan of treatment differs much from the procedure in a Type II case in which there is no overlapping. There is as much difference in the management of these two types as there is in the management of a catarrhal appendicitis and a ruptured gangrenous appendicitis.

(3) *Surroundings.* Is this patient to be home treated or hospital treated?

The psychology of the sick room is a factor for most patients; for an aged person it may well become one of the deciding factors. There are many spry old ladies of seventy who have never been under the care of a doctor except for a few days at each childbirth. They are made up of a combination of rubber and steel springs; they have been present at the birth and death of every grandchild; they are more active than their youngest daughter; they never in their lifetime weighed more than 120 lbs; they are the wrens of our practice as compared to the wise owls or old crows.

Put a patient of this perennial flapper type in bed and you by that act lower the morale to a visible degree. By contrast take the fat feeble septuagenarian who has led a sheltered life; who has avoided drafts; who has not ventured out in the night air; who has had a selected diet; who is constantly under the care of doctors. This patient is certainly not the wren type; no, that is a bird of another plumage or vintage—not infrequently a parrot. One of these two, with identical injuries, you treat like a doughboy; the other like a decrepit.

Let us be very careful then to treat the injured part in a way acceptable to the individual patient. Our splintage must not be cumbersome; we will avoid circular casts or casings; so far as possible we will apply only removable apparatus so that these aged tissues may be subjected to frequent inspection.

We will treat more of our fractures of the hip in traction and suspension and far fewer of them in abduction or other plaster-of-Paris casings or casts. To me it seems remarkable that so many of us long adhered to plaster-of-Paris spicas in fractured hips when most of us long ago discarded immobilizing dressings in every other joint because they kept the joint inactive so long and thus retarded circulation and delayed repair and convalescence. Yet the hip, more than any other joint, needs

conservation for there is a quadrilateral area of the neck of the femur which when broken never perfectly repairs. It is like the spinal cord in that respect. This is the area affected in the fractures of the base of the neck, the intracapsular variety. The others, those outside the base, the extracapsular, are in reality high fractures of the shaft of the bone with a prognosis more like shaft fractures.

I am convinced that Type 1 intracapsular fractures almost invariably repair by a sort of spontaneous reconstruction operation in which the fracture line fades out and the trochanteric line and the anatomical neck blend or fuse. The same thing happens with similar sort of bone as in the body of the vertebra, the os calcis, the lower end of the radius. It is a structural inability to repair; it is cancellous bone crushed and devitalized. In the neck of the femur this desiccated bone literally stews in its own juice (blood and serum and macerated muscle) in a vacuum under a pressure of many pounds at a temperature of nearly 100°F. Is it any wonder that under such conditions the neck disappears in some cases as early as six weeks after injury?

There are then certain rules and regulations governing the management of these aged injured, and of these let us not forget that one of the most important is to individualize our patient in terms of tissues rather than in terms of years.

This patient of seventy may be a better risk after an injury than that patient of forty-five.

And let us not be too prone to give a poor prognosis just because the patient is closer in years to one hundred than perhaps we ourselves will attain.

Some of these old models may not have four-wheel brakes, chromium plating, lacquer finish or rapid acceleration speed from a standing start; but just the same, this old model has gone many miles and is capable of going many more if not rushed into high speed too often.

THE OPERATIVE TREATMENT OF PARALYTIC DEFORMITIES OF THE FOOT*

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THE problem of the paralyzed foot differs radically from that of the paralyzed hand. In the hand our chief concern is restoration of the normal motion to each joint; in the foot, motion is of secondary concern; our chief aim is to establish stability. It is surprising how excellent the function of the foot may be with almost complete ankylosis of the superior and inferior ankle joints; with 15° of flexion and extension at the ankle, the limp may be so slight as to defy detection. This cardinal principle of stability, self-evident though it seems, is not yet generally accepted in foreign clinics, where the emphasis is still laid on restoration of motion to the paralyzed foot. In fact, to write autobiographically, I studied paralyzed feet and operated on them for eight years (from 1912 until 1920) before I realized this all-important truth. These years of study had, however, the great advantage of helping to perfect a technique of tendon transplantation which, when combined with adequate bone-stabilizing operations gives unusually gratifying results in dealing with paralyzed feet.

Historically the orthopedic profession has passed through somewhat the same phase of development as the author. In 1880 Nicoladoni attempted to restore function to a calcaneus deformity by transplanting the peroneal tendons. His operation succeeded technically, but failed to give adequate function. For many years, beginning in 1896 with Drobnick's emphasis on the importance of the periosteal implantation of the transplanted tendon, orthopedic surgeons vied with one another in the multiplicity, variety and ingenuity of tendon transplants. Lange in Munich is stated to have records of over 2000 such tendon operations. All these

operations aimed to restore motion to the foot; stability was to be gained by adequate restoration of muscle balance. In America too, the current of thought was similar. In 1913 the German and American currents met in the publication of an exhaustive treatise on tendons by Biesalski and Mayer. (*Die Physiologische Sehnenverphanzung*, Springer, Berlin.) This work contains valuable data on the anatomy and physiology of tendons, and details a painstakingly accurate operative technique for tendon transplantation. The word "stability" is, however, not once used in the entire volume; it is evident that the authors are thinking altogether of the restoration of muscle balance by the conservation of tendon function.

The importance of stability was first emphasized by Whitman in his astraglectomy and by Davis in his subastragloid arthrodesis. Too much credit cannot be given these pioneers. In 1922, after ten years of careful clinical experimentation, Dr. Michael Hoke presented an operation which, in its clear conception of the mechanics of the foot, in its precision and its adequate restoration of stability, constitutes to my mind the greatest single advance in the therapy of the paralyzed foot. Campbell and Putti have each contributed an important idea: Campbell in his posterior bone-block to prevent drop-foot, Putti in the anterior bone-block to prevent calcaneus.

With this brief historical review as introduction, I propose to outline the operative methods which I have employed during the past five years in the correction of the six main types of paralytic foot deformities: valgus, varus, equinus, calcaneus, cavus and dangle-foot. Most of these main types can be subdivided into

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different grades according to the degree of deformity and there are, of course, numerous sub-types representing combinations of the main deformities; for instance a calcaneo-cavus-varus, or an equino-cavus-varus. Each variety has its own special therapeutic indication and it is only by a careful pre-operative analysis that the appropriate therapy can be planned. Such statements as are frequently heard in orthopedic circles as "I favor bone operations" or "I have no use for tendon transplants," are meaningless since what is good for one type of foot is contraindicated for another.

PARALYTIC VALGUS

1. *Mild Grade.* Due to weakness of the tibialis anticus and posticus; no marked bony deformity.

Excellent results can be secured by manipulation (usually under anesthesia) and immobilization in a position of varus by a splint or, preferably, plaster-of-Paris. By cutting a window in the plaster, daily electrical stimulation with the sinusoidal current can be given the weakened muscles, which can be caused to contract, even though the foot itself does not move. After removing the plaster a calf-brace should be used to hold the foot in over-correction until the inverting muscles are strong enough to maintain muscle balance. When tested by the spring balance they ought, according to Osgood's statistics, to be slightly stronger than the evertors. An arch support should be worn if the valgus tendency is still present.

2. *Moderate Grade.* Paralysis of tibialis anticus with weakness of tibialis posticus and other inverting muscles. Good strength in extensor proprius hallucis and Achilles tendon. The weakness of the inverting group has caused definite bony deformity.

In this type of case I formerly secured fair results by transplanting the peroneus longus. During the past three years, however, I have found that the Hoke stabilizing operation gives excellent results without any tendon transplant. This is probably

due to the strong inverting force which can be developed by the synergistic action of the extensor proprius hallucis

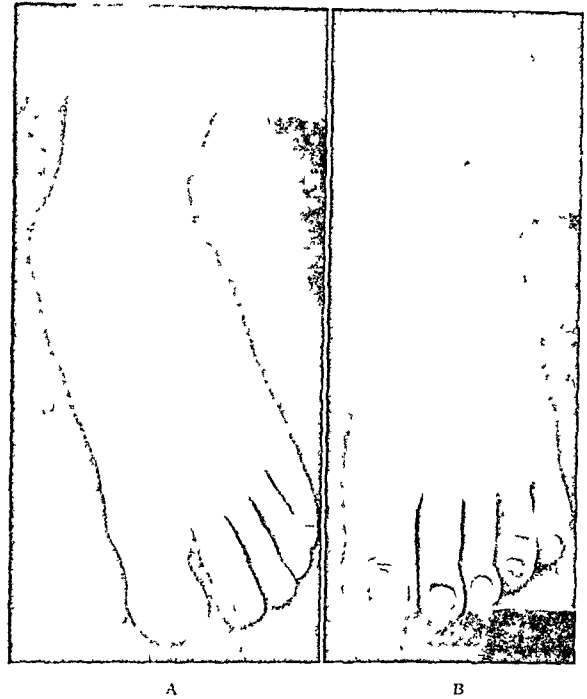


FIG. 1A. Paralytic flat-foot before operation. B. Paralytic flat-foot after operation. In this patient the Hoke stabilization operation was combined with the transplantation of the peroneus longus by the intrasheath method. The patient now walks without any limp.

and the Achilles tendon after the bony deformity has been corrected. Those who are interested in learning more about this striking muscle phenomenon should consult the chapter on muscle dynamics in the tendon monograph of Biesalski and Mayer. For the technique of the Hoke operation I refer the reader to Hoke's own accurate description. My one modification is the skin incision which I prefer making more vertical than Hoke is accustomed to, since this enables the operator to avoid cutting the superficial nerves and veins. As a rule patients operated on by this method require no support after several months of thorough postoperative treatment.

3. *Severe Grade.* Complete paralysis of tibialis anticus and posticus. Extensor hallucis proprius weak. Evertors strong; marked inward and downward displace-

ment of head of astragalus. The anterior tuberosity of the os calcis is depressed, making the inferior border of the os brevis spring from the distal half of the fibula; therefore to shift the tendon so as to change its everting action into inver-



FIG 2 A. Paralytic club-foot before operation B. Paralytic club-foot after operation. In this patient the Hoke stabilization was combined with the transplantation of the tibialis anticus to the outer side of the foot by the intrasheath method.

calcis cut the horizontal at an angle of 5° or less. (Fig. 1A and B.)

In this type neither tendon transplant nor bone operation is alone sufficient for permanent correction. This can, however, be secured by combining a stabilizing bone operation with the transplantation of one or more of the evertors. If the peroneus longus is strong it is the tendon of choice for transfer. Great care must be taken in accurate pre-operative muscle tests, since it is easy to confuse the peroneus longus and the brevis. The only accurate method is by palpation of the muscle belly during the act of contraction. If the peroneus longus is weaker than the other evertors (peroneus brevis, extensor longus digitorum and peroneus tertius) it is advisable to transplant the extensor digitorum and peroneus tertius, not the peroneus brevis. The reason is an anatomical one: the muscle fibers of the peroneus

sion, almost all the muscle fibers would have to be cut, thus destroying their contractile power. The extensor longus digitorum can be sacrificed without inconvenience to the patient, provided the extensor brevis digitorum is active. The distal stump of the extensor tendon to the little toe must, however, be spliced to the extensor brevis of the fourth toe, since the extensor brevis does not supply the little toe. For the complete technique of the tendon transplants the reader is referred to my previous publications.

In correcting the bony deformity I follow the Hoke procedure. I have found that in arthrodesing the calcaneo-astragaloid joint, I have occasionally over-corrected the eversion of the os calcis, and that the resulting inversion has seriously interfered with function, necessitating reoperation. This mistake must be carefully avoided. It is also important to restore

the normal inclination of the os calcis with the horizontal. This is best done by dropping the forefoot into a position of

greater or less degree. All these cases require bone operation. They may be divided therapeutically into two groups:



FIG. 3 A.

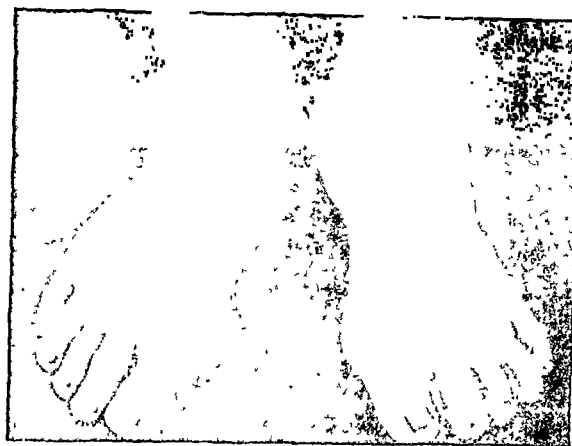


FIG. 3 B.

FIG. 3 A. Paralytic equino varus deformity before operation. B. Paralytic equino varus deformity after operation. A wedge resection from the astragalo-scaphoid and calcaneocuboid joints was done and in addition a lengthening of the Achilles tendon.

equinus, holding it there with a plaster mold, and then bringing the entire foot up to an angle of 90° . If the Achilles tendon is too short to permit this it should be tenotomized.

PARALYTIC VARUS

1. *Mild Grade.* Weakness of evertors without bony deformity.

Although cases of this kind can be held effectively by a brace, I advise operation, since a tendon transplant properly executed will give a complete cure. Either the tibialis anticus or the extensor proprius hallucis when shifted to the outer border of the foot by the intra-sheath method will produce adequate eversion. The tibialis anticus is the muscle of choice when a stronger action is required, first because it is more powerful than the extensor hallucis, second, because it is a stronger inverter whose loss as an inverter will make it easier to establish normal muscle balance.

2. *Moderate and Marked Grades.* Paralysis of evertors with bony deformity of

(a) Those in which there is either a tibialis anticus or extensor hallucis suitable for transplantation. (Figs. 2A and B.)

(b) Those in which these muscles are absent.

(a) The Hoke operation answers all demands for bony remodelling and stabilization. It is of importance not only to reshape the neck of the astragalus but to resect enough bone from the outer portion of the astragalo-calcaneal joint to permit complete correction of the inversion of the os calcis. In some instances the astragalus itself is inverted and its form must be corrected by the removal of a wedge from its body. These cases of varus, unlike the valgus, are not easily over-corrected. I can remember no instance in which this mistake occurred, whereas occasionally I have found the bony correction insufficient, and have re-operated to give additional correction.

After the bones have been re-shaped the tibialis anticus or the extensor proprius hallucis should be transplanted, preferably by the intra-sheath route. My only modification of the original technique is the method of fixation of the transplanted

tendon when the peroneus tertius is poorly developed; then it is better to drill a hole through the fourth metatarsal, and run

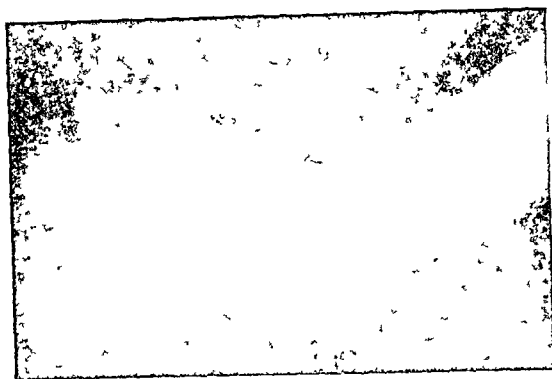


FIG 4. Paralytic drop-foot after the transplantation of both peroneal tendons to the dorsum of the foot. The peroneus longus was brought through the sheath of the tibialis anticus to the inner side of the foot, the brevis subcutaneously to the outer side. The picture illustrates the range of dorsiflexion by two exposures on the same plate.

the transplanted tendon through this bony channel by means of a long straight needle passed from the dorsum of the foot through to the sole.

(b) In the absence of the tibialis anticus or the extensor hallucis, the varus is almost always coupled with equinus deformity. This must be corrected by an appropriate lengthening of the Achilles tendon. (Figs. 3A, B.) To prevent return of the equinus, I combine the Hoke stabilization with the Campbell bone-block. Putti has recommended transplanting the tibialis posticus for this type of foot, running it through an opening in the interosseous membrane. Although my own experimental work would speak against the success of this method of transplant, I intend trying it when a suitable case occurs.

EQUINUS

It is surprising how well some individuals can walk despite complete paralysis of the dorsiflexors. These are usually patients with strong knee and hip muscles. Operation is indicated by an impairment of gait, not by the result of muscle tests. If the foot-drop is a definite source of diffi-

culty, two procedures are possible, depending upon the presence or absence of the peroneal muscles.

1. *Peroneals Present.* Excellent results are secured by the double transplantation of the peroneals; the longus is brought to the inner side of the foot by the intrasheath method, the brevis to the outer side by the subcutaneous route. (Fig. 4.) The intrasheath method is unfortunately not feasible for the brevis because of the following anatomical difficulties:

a. The divergence in the direction of the extensor longus digitorum sheath and the direction of the transplanted tendon.

b. The nature of the intermuscular septum separating the peroneals from the anterior muscles.

c. The low origin of the muscle fibers of the peroneus brevis.

Despite these difficulties the brevis can be made to act as a dorsi-flexor though not as effectively as the longus.

In addition to the tendon transplants a Hoke stabilization should be done to lessen the possibility of a varus deformity which might develop after shifting the peroneus longus to the inner side of the foot.

2. *Peroneals Absent.* There are many more cases in this group than in the first. Since there are no muscles available for transplantation, recourse must be had to a bone-blocking operation. The method of Willis Campbell is in my experience the best. I have tried to modify his technique by using a single large tibial graft instead of a number of small grafts, but I have found his original method more satisfactory. The results are good, provided the foot is kept at an angle of 90° for at least four months after the operation. It will then drop about 10° giving it a useful range for the average stride.

CALCANEUS

Owing to the paralysis of the gastrocnemius and soleus, the normal upward pull on the posterior tubercle of the os calcis is lost, and consequently the os calcis is tilted by the pull of the plantar muscles,

until its axis becomes increasingly vertical. The gait in calcaneus is always poor.

It was for the correction of this deformity

Since the Putti bone-block has thus far not been adequately described in the American or English literature, I feel

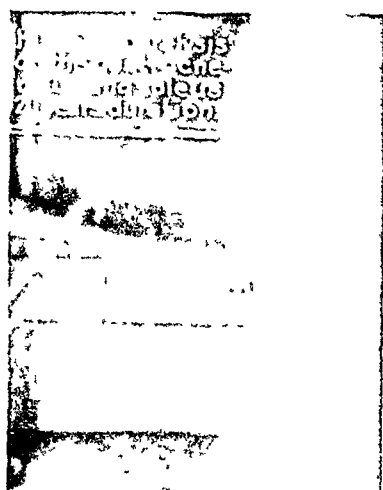


FIG. 5 A.

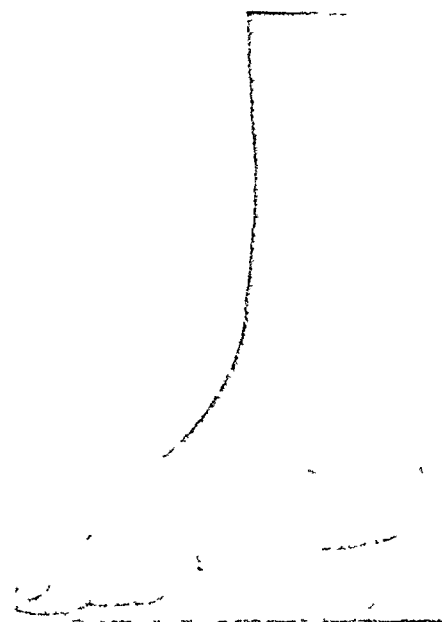


FIG. 5 B.

that Whitman devised the operation of astraglectomy, and it is in cases of this kind that his operation gives its best results. I am, however, opposed to the routine use of astraglectomy to correct unilateral calcaneus for the following reasons:

a. It shortens the leg from $\frac{1}{2}$ in. to $1\frac{1}{4}$ in. depending upon the age of the patient.

b. Esthetically the appearance of the foot, even after successful operations, is not as pleasing as after other operative procedures.

c. Frequently after a number of years there develops an adduction of the forefoot.

d. It is difficult for patients with astraglectomy to walk barefoot and to go up grade.

Despite these disadvantages the operation must be hailed as the pioneer of all our stabilizing procedures. It unquestionably corrects the disfunction of calcaneus. My personal experience, however, is that the Hoke operation modified by the Putti bone-block and combined with effective tendon transplantation gives superior results.



FIG. 5 C.

FIG. 5 A. Calcaneus deformity before operation. B and C. Calcaneus deformity after operation. Head of the astragalus was resected and ligaments of the os calcis freely divided to permit posterior displacement. Transplantation of both peroneals and flexor longus hallucis to replace paralyzed Achilles tendon.

warranted in giving its details: An oblique incision over the external aspect of the

ankle exposes the upper ankle joint. The tendons, vessels and anterior tibial nerve are retracted inward, until the upper sur-

ough division of both the long and short plantar ligaments and of the plantar fascia, permits correction of the cavus.

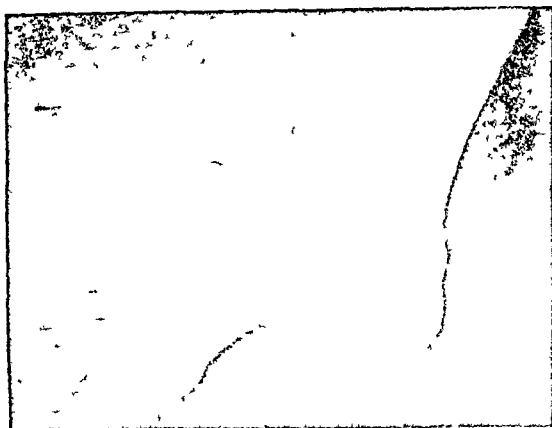


FIG. 6 A.

face of the body of the astragalus is in full view. The foot is then tilted downward, so as to bring the posterior portion of the astragalus into a position which would correspond to about 110° of equinus. A thin chisel is then driven vertically into the body of the astragalus, splitting it for the reception of a tibial bonegraft measuring in width a trifle less than the astragalus, having the thickness of the tibial cortex and long enough to project $\frac{3}{8}$ in. beyond the astragalus. This graft, healing firmly with the astragalus, effectively blocks excessive dorsiflexion and at the same time permits sufficient motion for a good stride. My earliest case operated on in 1924 still has an effective block, without any irritative symptoms. Putti has followed some of his cases for ten years without noting any untoward results.

Putti's procedure does not attempt correction of the cavus, nor does it give the posterior displacement of the os calcis which is so important a feature of Whitman's operation. To accomplish this double purpose I have modified the Hoke stabilization: Instead of shortening the neck of the astragalus as he does, I resect enough of the head to permit generous posterior displacement of the os calcis after division of all the astragalocalcaneæ ligaments. The additional resection of bone at the calcaneo-cuboid joint, together with thor-

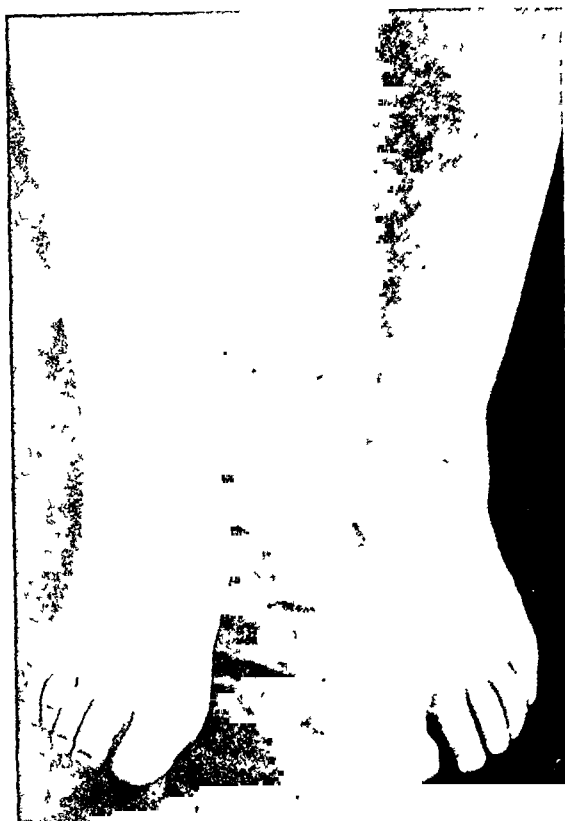


FIG. 6 B.

FIG. 6 A. Pes cavus before operation. B. Pes cavus after operation. In this patient a wedge with the base directed towards the dorsum of the foot was removed through the Chopart's joint and the plantar fascia and muscles were freely divided.

For tendon transplantation there are available both the peroneals, the tibialis posticus and flexor longus hallucis (the flexor longus digitorum is too small a muscle to warrant transplanting). At least two tendons should be transplanted, one from the outer group, one from the inner; and I have frequently taken three. They are all brought through a slit in the Achilles tendon and fastened to the os calcis near its posterior tubercle. The results have given good function without the use of apparatus. (Figs. 5A, B, C.)

CAVUS

Paralytic cavus is usually easier to cure than the idiopathic type. The latter tends to recur after an apparently successful

operation; the paralytic does not, provided the bony deformity has been completely overcome.

by Hibbs. The tendons are cut at the level of the metatarsal heads, dissected upward almost to the ankle, swung to the outer

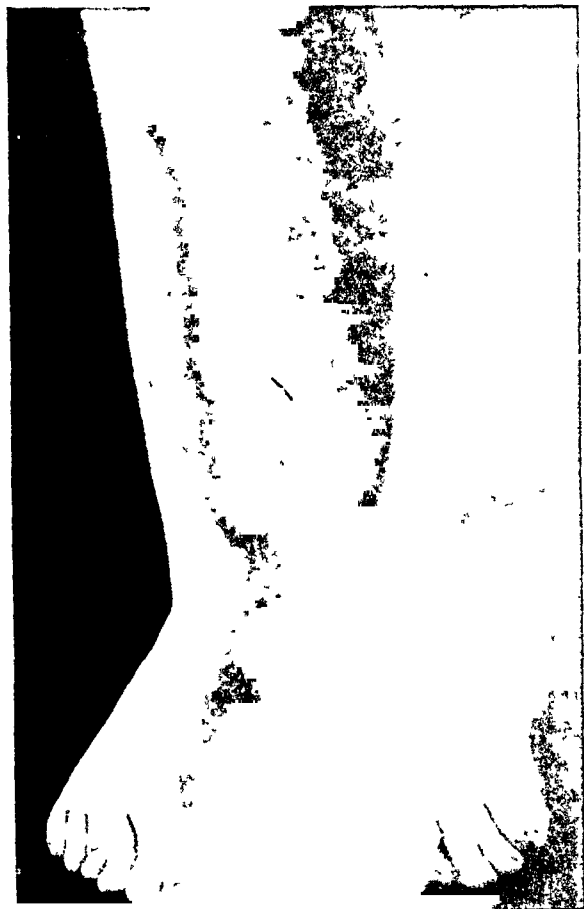


FIG. 7 A.

Cases can be divided into the mild type and the more marked grades. In both there is usually a complicating hammer position of the toes, due to the sharp downward inclination of the metatarsal bones.

1. *Mild Grade.* There is little or no bony deformity. The chief obstacle to correction is the shortening of the plantar fascia, muscles and ligaments.

In these feet the Steindlar stripping operation is effective. As a rule the deformity of the toes requires either tenotomy of the extensor tendons or their implantation into the metatarsal heads. If there is a complicating tendency to inversion, I transplant the extensor longus digitorum to the outer side of the foot in somewhat the same way as in an operation described

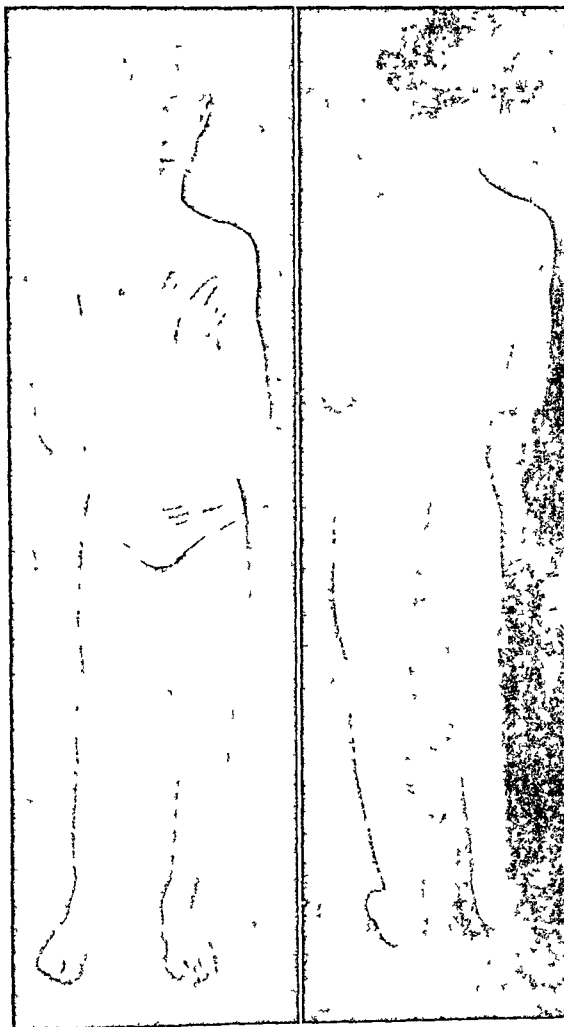


FIG. 7 B.

FIG. 7 A. Right side dangle-foot, left side paralytic valgus (before operation). B. Right side dangle-foot, left side paralytic valgus (after operation). Right foot: a Hoke stabilization was combined with partial removal of cartilage from the astragalotibial joint. Left foot: a Hoke stabilization was combined with transplantation of the peroneus longus by the intrasheath method.

side and passed through a drill hole in the fifth metatarsal.

2. *Severe Grade.* There is bony deformity of greater or less degree.

Under these conditions the Steindlar operation alone has not been successful in my hands, but a bone operation has been. A bone wedge with its base toward the

dorsum is taken through the calcaneo-cuboid and astragaloscaphoid joints. The more marked the cavus, the broader the wedge. The long and short plantar ligaments are divided beneath the cuboid bone and the plantar fascia is divided subcutaneously. Correction of the deformity should then be easy. The toes are dealt with as in the mild type. (Figs. 6A, B.)

DANGLE FOOT

Under this term I include those feet which are practically without muscle power. The therapeutic grouping depends upon the condition of the knee and hip muscles.

1. *Knee and Hip Muscles Functioning.* Under these conditions excellent function can be secured without ankylosing the ankle. If some motion can be conserved and at the same time stability maintained, the gait will be better than if the ankle joint is firmly arthrodesed.

I have followed two methods to secure this result. The first is simpler, the second, though more difficult, is more accurate. In both the operation is begun by arthrodesing the astragaloscaphoid, calcaneo-cuboid and calcaneo-astragaloid joints. In the first, the tibiotarsal joint is then opened and part of the cartilage is scraped away from the articulating surfaces of the tibia and astragalus. Some cartilage is left, so as to insure the persistence of a little motion in the upper ankle joint.

(Figs. 7A, B.) In the second the Putti bone-block is inserted into the astragalus anteriorly, and the Campbell bone-block posteriorly, allowing about 15° of flexion and extension. Although this method takes longer, the results have been particularly gratifying.

2. *Knee and Hip Muscles Weak.* In this type it is desirable to secure complete ankylosis of the entire ankle joint. To insure this I not only denude the joint of cartilage, but run a tibial bone graft from the tibia into the body of the astragalus. The subastragaloid, the astragaloscaphoid and calcaneo-cuboid are arthrodesed in addition.

SUMMARY

Paralytic deformities of the foot are almost without exception amenable to operative correction. The aim of the orthopedic surgeon is the construction of a stable, well-balanced foot of good esthetic form, capable of function without a brace, and adapted to the wearing of a ready-made shoe. To secure this result a rigorous pre-operative study of each foot is necessary. The operative indications must be made to conform to the pathological findings, the operative technique must include adequate skill in tendon transplantation as well as in bone remodelling, and the postoperative treatment must be carried through until the correction has been made permanent.

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NOTE: For remainder of bibliographic items see author's reprints.

SCIATIC SCLIOSIS DUE TO LOW BACKACHE*

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SCIATIC scoliosis is a painful lateral deformity of the trunk which affects only adults. The name is not descriptive of the actual condition because there is no real scoliosis, and pain along the great sciatic nerve is an inconstant symptom. However, the term sciatic scoliosis has been used for many years and is universally understood to apply to a definite clinical picture. This clinical picture or group of symptoms may complicate or occur in the course of any one of a variety of orthopedic pathological conditions in the lower part of the back.

The chief symptoms of sciatic scoliosis are a lateral deformity of the trunk, low backache, stiffness of the back, disability and frequently radiation of the pain down one lower limb, accompanied sometimes by atrophy of some of the thigh muscles and the buttock. The trunk is always inclined laterally and a little forward. In the majority of cases the trunk is tilted toward the sound side. The degree of deformity varies in direct proportion to the severity of the pain. In the mild cases there may be only slight lateral distortion, which may be readily corrected voluntarily, and which always disappears when the patient sits or lies down. In the more advanced cases the deformity cannot be reduced voluntarily and persists when the individual lies down. The deformity varies also in degree at different stages of the affection, but it is always severe when the pain is marked. The body usually remains tilted to the same side, but in a small per cent of the cases there is an alternating deformity; that is, in a case of chronic sprain of the right sacroiliac joint, for example, the body may be bent to the left side one day and to the right side on the following day.

The deformity of the vertebral column in this condition coincides with that of the trunk. In a right sciatic scoliosis the dorsal and upper lumbar vertebrae are deviated to the right, while the lower lumbar vertebrae curve to the left. There is also a marked change in the anteroposterior curvature of the spine. The normal lumbar hollow or lordosis is either reduced or obliterated, or it may be replaced by a backward curve or kyphosis of the lumbar vertebrae. The dorsal region, which normally is rounded and curves backward, becomes flat. The appearance of the back as a whole is that of unusual flatness. There is spasm of the back muscles and marked restriction of all the motions of the spine, resulting in stiffness of the back. The motions of the spine are not equally limited. Flexion is usually more markedly restricted and more painful than the other motions.

The pain is located in the lower part of the back. Depending on the exact pathology, it may be most marked over one of the sacroiliac joints, the lumbosacral junction, the musculature of the back or the gluteal region, and frequently it radiates down the back of the thigh and leg on the affected side. The tenderness, which is usually present over the painful areas, is more marked over a joint than over soft tissues such as the muscles or along the sciatic nerve. The pain is increased when the sensitive tissues are stretched. Thus in a derangement of a sacroiliac joint or myositis of the gluteal area the pain is aggravated by flexing the trunk or elevating either lower limb with the knee extended, because these motions result in tension on the gluteal muscles and sacroiliac joints. The pain is also increased by all motions of

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the trunk. Consequently lying down, turning in bed, getting up from the recumbent or sitting position and any other change of position of the body is very painful. At times the pain is so severe that the patient dreads even the slightest movement of his body. Pain along the back of the thigh and leg, that is, the so-called sciatic pain, may be the only pain present, but it is usually coexistent with pain in the back. The pain along the sciatic nerve is responsible for the name sciatic scoliosis, since originally it was believed that there was a primary sciatica and a secondary scoliotic deformity. The reflexes are not altered, but there is frequently a marked degree of atrophy of the buttock and thigh.

ETIOLOGY AND PATHOLOGY

The deformity of sciatic scoliosis is the result of an automatic attempt by the patient to relieve himself of pain in the back or along either lower limb. Thus we find that sciatic scoliosis may result from lesions in the following parts:

1. The sacroiliac joints
2. The lumbar vertebral joints
3. The muscles and fascia of the back
4. The gluteal tissues
5. The sciatic nerves

When there is pain in any of these areas the individual instinctively shifts his body until he finds a position in which there is the least discomfort, and then he maintains that position by strong muscular spasm.

Derangement of the Sacroiliac Joint. The most frequent lesion of the sacroiliac joint causing sciatic scoliosis is a sprain. This may be brought on by a very slight injury such as straightening up from the stooped position, an attempt to lift an object or a more severe trauma, as in a fall. It is scarcely ever the result of a direct blow. The lesion results, I believe, from an unbalanced or uncoordinated sudden contraction of the muscles of the back and pelvis. The sacroiliac ligaments are strained and a synovitis follows. The sprain may

be only momentary, or it may persist for hours, days or weeks. The symptoms may disappear and recur at irregular intervals with or without additional injury, or there may result a chronic persistent disabling deformity. A succession of strains is likely to cause an arthritis of the sacroiliac joint. Dr. Smith-Peterson of Boston has brought convincing histological proof that at least in some cases there is a distinct arthritis. I myself have seen at operation several sacroiliac joints in which there was great irregularity of the joint surfaces and of the articular cartilages.

The direct clinical evidences of the existence of a sacroiliac disturbance are pain and tenderness localized at the affected joint, and pain in the sacroiliac joint on straight leg raising. The roentgenograms are of but little help. In most cases they are entirely negative. Occasionally there is haziness of the joint suggesting a possible intra-articular effusion or peri-articular infiltration. Sometimes there is an inequality in the levels of the joint surfaces of the ilium and sacrum, which has been interpreted as a subluxation of the joint. Consequently, if an individual after an injury complains of pain in one sacroiliac joint, has the deformity of sciatic scoliosis, and the joint is sensitive to pressure, we conclude that he has a sciatic scoliosis secondary to a derangement of the sacroiliac joint.

Lesions of the Lumbar Vertebral Joints. Similarly an injury may cause a sprain of the lumbosacral or of one or more of the other lumbar vertebral joints. A focus of infection in a tooth, the tonsils, sinuses or the colon may result in a mild arthritis or peri-arthritis of the lumbar joints. The infection may subside, but it may leave adhesions about the vertebrae causing persistence of the deformity. The changes in the lumbar spine are evidenced by tenderness over the lumbar joints and by restricted mobility. The roentgenograms have a negative value in indicating an absence of serious bone damage. Sprain of the lumbosacral joint is, in my experi-

ence, nearly as common as that of the sacroiliac joint. Frequently they occur simultaneously. In a lumbosacral sprain the pain and tenderness are localized just above the sacrum and the spinal motions are restricted in all positions of the trunk. Thus, while in a sacroiliac derangement, flexion of the spine is restricted when the patient is standing, and fairly free when he is sitting, in a sprain of the lumbosacral joint the restriction is equally marked in both positions.

Lesions of the Muscles and Fascia of the Back. An injury may cause a strain of the muscles and other soft tissues of the back. This condition corresponds to the old-fashioned lumbago and consists of an injury to some of the muscle fibers by a sudden or unexpected motion. There is probably a tear of some of the muscle fibers with extravasation of blood into the muscles and a hyperemia. The patient suffers a variable amount of pain which is increased by all motions. There is tenderness of the muscles. In the chronic cases there may be adhesions between the spinal muscles causing the pain and restriction of spinal mobility. The latter is an inference based on the observation that in the chronic cases stretching of the spine under an anesthetic gives complete and permanent relief. In view of the reports of Dr. Albee and Dr. Osgood it is likely that, at times at least, there is a systemic toxemia from some focus in the intestines and a resultant myositis of the muscles of the back causing sciatic scoliosis which, fortunately, is curable by colonic irrigations and dietary regulations.

Gluteal Myositis. In some cases of sciatic scoliosis the patient complain of pain only in the buttock. There is usually tenderness of the gluteus medius and of the tissues attached to the posterior surface of the sacroiliac area. There are no palpable masses, local heat or enlarged lymphatic glands, that is, no evidence of a true inflammation. The condition is probably due either to an injury or a toxic condition

of the gluteal muscles. Dr. Percy Roberts of this city called attention to this condition a number of years ago, and recently, Dr. Albee has emphasized the importance of this lesion in a paper on myofascitis.

Sciatica. We must consider also a group of cases with the typical deformity of sciatic scoliosis in which the patients complain only of pain along the back of the thigh and leg, that is, along the sciatic nerve. In these patients there is tenderness in some spots along the course of the sciatic nerve, particularly at the great sacro-sciatic notch or exit of the nerve from the pelvis. There may be some muscle atrophy in the thigh and buttock. There are no changes in the reflexes, no trophic changes in the skin and there is no weakness of any of the muscles. Therefore, the cardinal symptoms of a sciatic neuritis are wanting, but for convenience we speak of the condition as a sciatic neuralgia or sciatica. Its exact etiology and pathology are not understood. Formerly the neurologists used to think there was a primary disturbance of the sciatic nerve. This is very doubtful and now most observers believe that the sciatica is secondary to a lesion of the joints or muscles in the lower back. Dr. Mark Rogers in an analysis of 50 cases of sciatica found an arthritis in the lumbar or sacroiliac articulations in 49 of them. In the remaining case the pain was due to a carcinoma of the prostate. The sciatica is in these cases probably due to pressure upon the lumbosacral cord and branches of the sacral plexus as they pass over the inflamed or otherwise deranged last lumbar and sacroiliac articulations.

PROGNOSIS AND TREATMENT

The prognosis of sciatic scoliosis resulting from the various conditions we have discussed is exceedingly favorable, and one may confidently expect a cure with treatment. For purposes of convenience one may speak of three degrees of sciatic scoliosis, mild, moderate and severe, depending upon the intensity and duration

of the deformity. In the mild form the patient has some pain, and may or may not be aware of the distortion of the trunk.

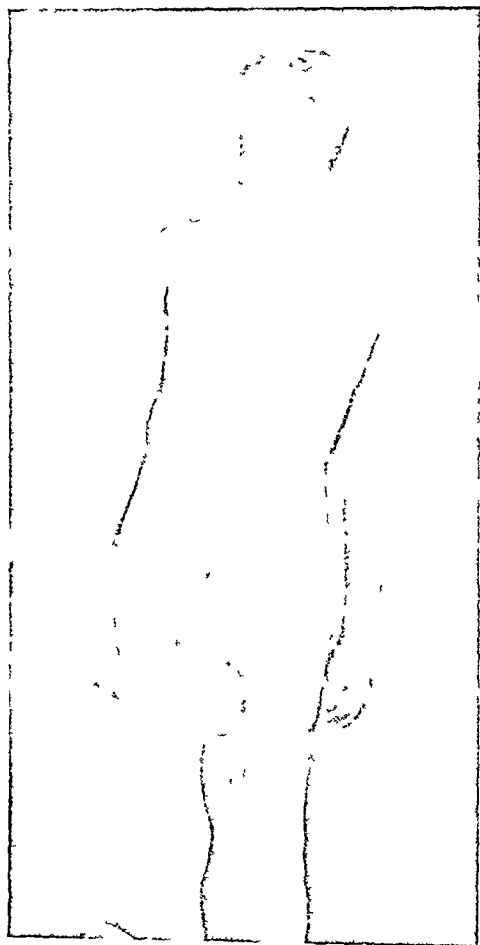


FIG. 1. Right sciatic scoliosis secondary to right sciatica. Typical deformity of sciatic scoliosis: forward and lateral deviation of trunk; flatness of back and evident stiffness of spine. Dorsal area of spine deviated to right, and lower lumbar area to left.

Because of the pain he limits his activity and in a short time the symptoms disappear.

In the moderate cases the symptoms are more pronounced and lasting. The treatment can usually be effectively applied in the patient's home or in one's office. If the joints are affected one applies support by means of adhesive plaster or flannel bandages to immobilize the joint. Physiotherapy in the form of baking, diathermy and massage give relief from pain. Rest in bed for several days

may bring complete relief. Removal of evident foci of infection and colonic irrigations have in some cases yielded surpris-

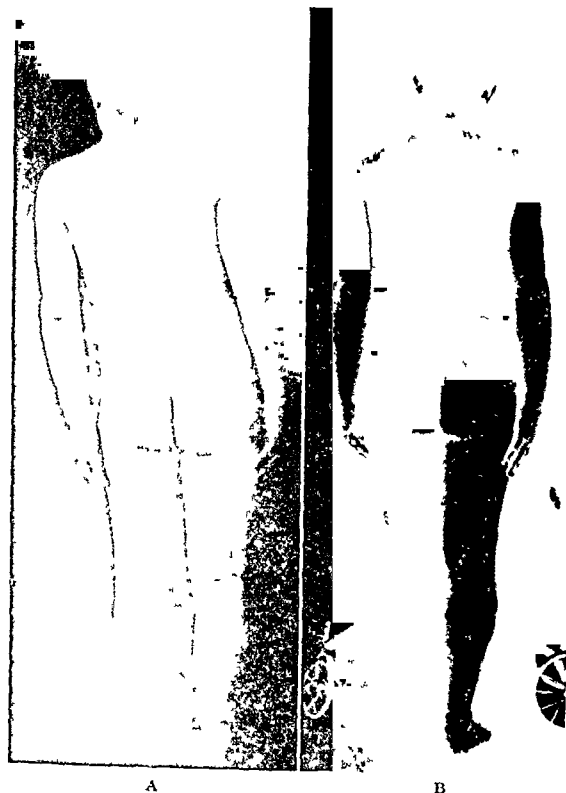


FIG. 2 A. Left sciatic scoliosis due to strain of right sacroiliac joint. Duration fourteen weeks. Typical deformity with lateral deviation of trunk to sound side. B. Same patient after stretching. Deformity corrected, back symmetrical and patient free from symptoms.

ingly rapid cures. In sciatica and gluteal myalgia the actual cautery may give greater relief than any other procedure. If the pain is very disturbing analgesics may be necessary for a short while. Epidural and perineural injections of novocaine or even normal saline solution have given relief in some cases. Ordinarily the symptoms gradually subside and disappear in several weeks.

In severe or resistant sciatic scoliosis in which the suffering is intense or has been prolonged, and in which the deformity is marked and more or less completely disabling, hospitalization and stretching of the back or sciatic nerve or both under an anesthetic constitute, in my experience, the shortest and surest method of obtaining

a complete cure. In such cases the deformity has become confirmed either through adhesions or accommodative changes in the soft parts. The adhesions must be broken up and the muscles stretched. Accordingly the patient is given an anesthetic and placed on an operating table of the Hawley or Albee variety. The procedure then differs according to the underlying lesion. If the pathology is in the back or sacroiliac area, the spine is bent forcibly from side to side and the legs are brought down into marked hyperextension. In this act the adhesions are torn, the sacroiliac joint is brought to a normal state and the normal lumbar hollow is re-established. After padding the trunk thoroughly and placing a very thick pad of felt or cotton over the lumbar area to maintain the lumbar hollow, a plaster-of-Paris jacket is applied. When the plaster has hardened sufficiently the legs are brought up to normal alignment with the trunk, and the plaster dressing is continued down the leg on the affected side to the toes. The completed splint is a plaster-of-Paris spica jacket extending from the nipple line to the toes. If the chief lesion is a sciatica the procedure consists of a stretching of the sciatic nerve. This is accomplished by bringing the leg forward, with the knee in complete extension. The limb is gradually but persistently flexed on the abdomen until the foot is brought within 15 or 18 in. of the face. This must be done slowly and cautiously. It should take no less than five, but preferably ten minutes. As the limb is brought forward the sciatic nerve is stretched and the adhesions about it are torn. The limb is then brought to a normal alignment with the body and a plaster-of-Paris spica jacket is applied. This procedure has one element of danger, namely, that too vigorous stretching may cause sciatic paralysis. I mention this because I have heard of such cases, though fortunately I have had no such personal experience.

A truly remarkable feature of this stretching of the back and the leg is that,

when the patient awakens from the anesthetic, he has lost all his old pain. This is, I believe, due solely to the stretching of contracted muscles and release of adhesions. The plaster is left on for about a month. It is then removed and active therapy is begun. This is a very important period, as the result may be upset by injudicious enthusiasm. The patient is not ready to go back to work as soon as the plaster is removed. He is first measured for a Knight spinal brace to continue the support of the back. While this is being made, during the course of a week or ten days, the patient is given physiotherapy twice a day in bed. Several times a day he exercises his leg and back muscles. One begins with a few exercises occupying about five minutes, and increases these daily so that at the end of ten days the patient can sit up with his trunk entirely symmetrical. The spinal brace is then applied and the patient permitted out of bed. Within another week he is walking about freely and can go home. He then spends another two weeks exercising more extensively and soon he returns to his work. The brace should be worn during the day for several months and laborious work should not be undertaken for at least three months.

The routine above outlined is varied, of course, depending upon the physical make-up and sensitiveness of the individual. Sometimes it takes six months to obtain a complete cure; at other times the patient is ready for a fairly hard day's work at the end of two months. As an encouragement to those who must advise such patients, I want to say that I have never seen a case of sciatic scoliosis due to a derangement of the joints or muscles of the lower back, and not due to a destructive disease or malignant bone lesion, that did not respond satisfactorily to the treatment as described. Our greatest difficulty arises in convincing patients, and sometimes doctors, of the advisability of hospitalization.

SUMMARY

Sciatic scoliosis then is a deformity of the trunk resulting from usually a traumatic, sometimes a toxic, condition of the joints or soft tissues in the lower back, myositis or myofascitis of the gluteal region, or sciatica. The mild and moderate cases can be relieved by simple

measures such as rest, support of the back, external heat, massage, diathermy or the use of the cautery. The severe and chronic cases can be cured by stretching the back and affected limb under an anesthetic, immobilization in a plaster-of-Paris spica jacket and rest in bed for about a month followed by a brief period of graduated exercises and physiotherapy.

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A COMBINED SUPERFICIAL AND DEEP CONTINUOUS SUTURE*

A NEW HEMOSTATIC, APPROXIMATION & TENSION SUTURE

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THE proper suturing of wounds plays an important rôle in the process of healing. Postoperative wound infections in clean cases often may be attributed to either too lengthy operations, imperfect hemostasis or improper approximation of cut edges, especially the skin. A prolonged operation saps the patient's vitality and exposes the wound for a longer time to possible contamination. It thereby lessens the individual's resistance and increases the chances of infection. Imperfect hemostasis may cause a hematoma which tends to separate the skin edges and leads to infection. Uneven approximation at the line of incision, especially the skin, predisposes to supuration and the formation of exuberant granulations. It results in tardy healing and an ugly scar.

Sir Berkeley Moynihan, in discussing intestinal obstructions, writes: "During the operation, the surgeon will need all his dexterity, rapidity and judgement. In all abdominal operations speed is a desirable thing; here it is an imperative necessity. The surgeon must discover what has to be done and do it with all dispatch." The first desideratum, then, is reasonable operative speed without sacrificing thoroughness. This is best attained by the acquisition of anatomical knowledge and practical experience in addition to one's natural dexterity.

It is not the purpose of this paper to enter into any phase of this discussion. However, I should like to suggest an aid in the matter of hemostasis and proper approximation of wound edges, by the use of a new suture to be described.

No doubt we are all aware of the great

variety of methods of suturing used to approximate skin edges. The continuous suture, the interrupted, the lock stitch and the figure-of-eight sutures are but a few of the many in vogue. One therefore feels that there is very little room for any additional method of suturing. Such was my opinion until I struck upon the suture to be described. It appeared to be ideal from many viewpoints and far superior to any of the existing methods. I should like to describe briefly this simple suture.

In suturing the skin, one should endeavor to obtain a perfect, even and firm approximation so as to leave as fine a scar as possible with the least tendency for the scar to stretch. Also, one must obtain perfect hemostasis of the skin and subcutaneous tissues so as to avoid secondary bleeding which often results in infection and breaking down of the wound.

In using the sutures thus far adopted these desirable requisites are not always readily attained. If one uses a *superficial* skin suture very close to the skin edge, whether it be continuous or interrupted, there is a tendency for the skin edges to pull apart and separate between the stitches as there is very little approximation of the raw surfaces underneath the dermis (Fig. 1). There is also a tendency to capillary oozing from the deeper parts of the skin incision.

If one uses a *deep* skin suture quite a distance away from the edges one partly obviates the tendency to bleeding and separation of the skin edges but finds himself confronted with either inversion or eversion of the skin edges with pouting of the subcutaneous tissues resulting in poor approximation and ugly scars.

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The suture to be described eliminates these difficulties. It is most simple in execution. It is similar to the ordinary

is repeated. In this new suture (Fig. 2), instead of carrying the needle over the line of the skin incision to the opposite

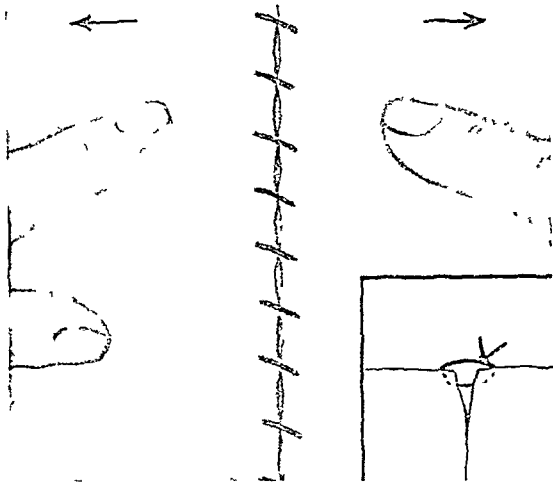


FIG. 1. Ordinary continuous suture. Note gaping and separation of skin edges between sutures that may take place if tension is applied to them.

continuous stitch with the following exception: in the ordinary continuous stitch (Fig. 1), one passes the needle through the skin a distance away from the skin edge on one side until it reaches the opposite

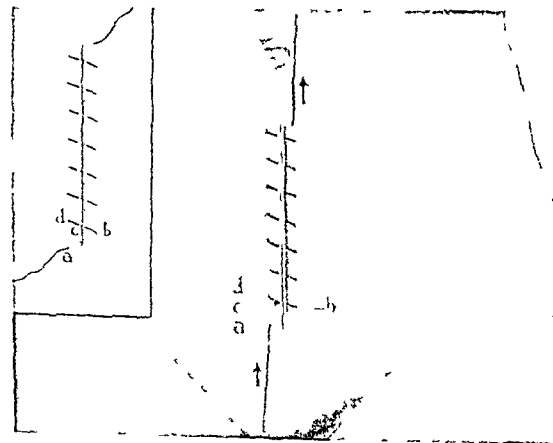


FIG. 2. Combined superficial and deep continuous suture. Needle enters skin at a, passes through skin and subcutaneous tissues, comes out at b, passes through skin edges at c, then goes to d, from where same process is repeated.

side of the incision, the same distance away from the skin edge. Then the thread with the needle is carried over the line of incision in an oblique or straight direction to the opposite side, and the same process

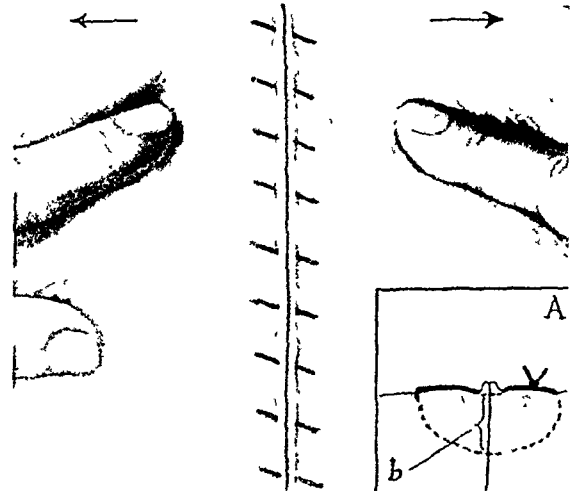


FIG. 3. Combined superficial and deep continuous suture. Note firm and even approximation at line of incision with no tendency toward separation even when tension is applied to it. Insert A: Note wide and close approximation of raw surfaces of skin and subcutaneous tissues b brought about by use of this suture. Compare with Figure 1.

side as mentioned above, the needle as it reaches the extreme edge of the skin

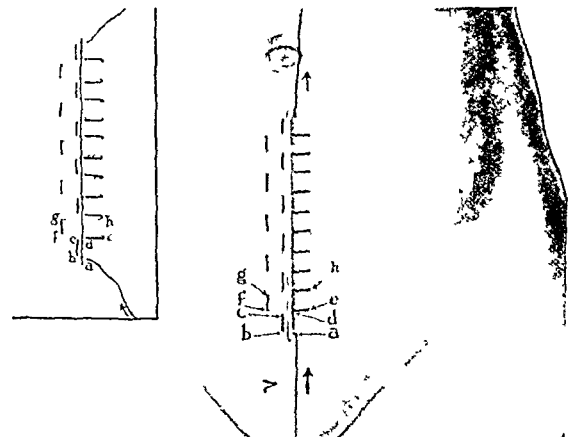


FIG. 4. Combined superficial and deep continuous mattress suture. Needle enters through skin at a, passes through skin edges to b, goes to c, passes through skin edges to d, goes to e, passes through a good thickness of skin to f, goes to g, and passes through a good thickness of skin to h. From there needle again enters as in a and steps are repeated until wound is closed. We thus have one superficial mattress stitch alternating with a deep mattress stitch.

at the line of incision is first passed through

these free skin edges and is then continued as in the ordinary stitch.

What has been accomplished? By pass-

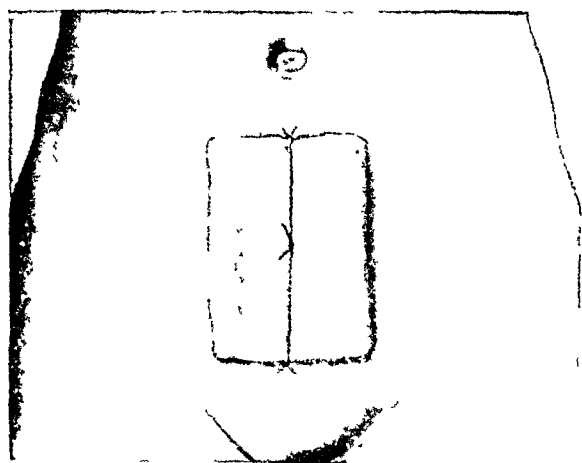


FIG. 5. Continuous suture tied over a gauze pad.

ing the needle through the extreme edges to either side of the line of incision, it approximates them and prevents the edges of the skin from either everting or inverting (c) and by passing the needle with the

a superficial and a deep bite of the skin (Fig. 4).

One thus gets the benefit derived from the superficial line of sutures which spells close and even approximation and the deep line of suture which aids in bringing together a greater raw surface for approximation, prevents capillary oozing and takes off the tension from the superficial line of suture thus preventing the scar from stretching (Fig. 3). This forms a hairline scar at the site of approximation of the skin edges.

This one suture is in reality a combination of a continuous approximation and a relaxation suture without using any additional suture material. It is most applicable in the approximation of skin edges. Its principle may at times be applied to other structures such as peritoneum, mucous membrane, muscle or fascia. This may be used in the peritonealization following hysterectomy, the suturing of the anterior or posterior vaginal wall

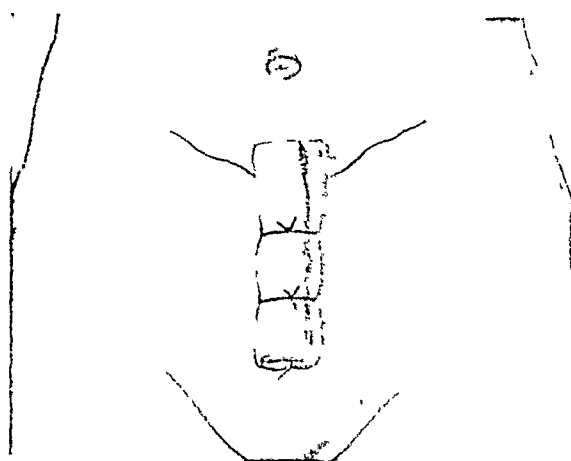


FIG. 6. Gauze pad folded over continuous suture. Two silk worm figure-of-eight sutures are tied over folded pad while one is still to be tied.

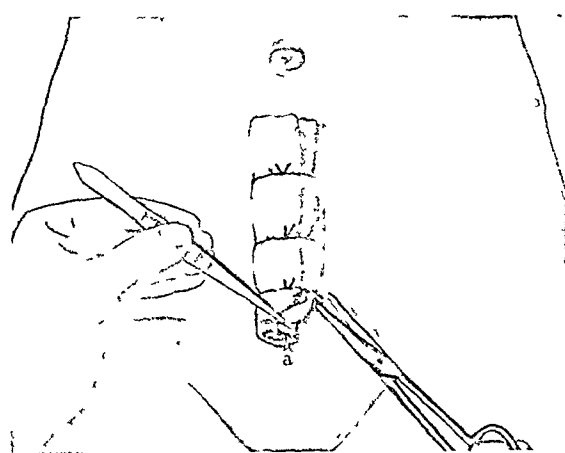


FIG. 7. Continuous and interrupted sutures ready to be removed. Gauze pad is lifted up at one corner by forceps while scissors depress skin near silk worm figure-of-eight suture to be removed. Suture is thus brought into strong relief and is easily cut. Continuous suture is then cut at lower angle a in a similar manner. Additional sutures are removed by same process.

thread through the deeper parts of the incision (a, b), one gets a stronger and wider approximation at the line of incision and takes off the tension from the skin edges through which the superficial part of the suture is passed (Fig. 3). The same principle may be applied when using a continuous mattress suture by alternating

during colporrhaphy or the approximation of conjoined tendon to Poupart's ligament in hernia operations. Once this suture is tried, one cannot help but be impressed with its simplicity and effectiveness as

well as the ease with which it is removed. The accompanying illustrations will enable one to visualize its practicability and simplicity.

An ideal closure and dressing for clean wounds, especially in abdominal surgery, is the use of the suture above described in combination with the ordinary silkworm figure-of-eight interrupted suture for skin and fascia used in the following manner: A small pad of gauze is placed over the continuous suture and the loose ends of the silk are tied over this gauze pad (Fig. 5). The edges of the gauze pad are then folded over the tied silk suture and the silkworm sutures are then tied over the pad at right angles (Fig. 6). We thus have a fixed dressing which cannot fall off, the pad preventing the figure-of-eight sutures from cutting into the skin. It acts as a bolster and by its pressure it also acts as a miniature sand bag to avoid dead space between the skin and fascia. Should the remaining dressings, including the adhesive, etc., be accidentally removed by a restless patient, there is still enough of a dressing left temporarily to prevent the wound from becoming infected. Likewise, the interrupted sutures are more easily

removed when necessary by lifting the gauze pad away from the skin. This in turn brings into relief the silkworm strands as well as the silk ends (Fig. 7). One thus easily cuts the sutures at one side and lifts them up together with the gauze pad.

The writer has used this method of closure in almost all clean cases on his surgical service both at the United Israel-Zion Hospital and the Brownsville and East New York Hospital. Since its adoption, about three months ago, it has been used in over 300 cases with the most gratifying results. All the wounds healed by primary union and the line of suture could hardly been seen if at all. It leaves a hairline scar surely no thicker than a crease line in the palm of the hand.

The simplicity and ease with which this suture is passed; the automatic hemostasis and even approximation of the wound edges which occurs when the thread is pulled upon; the resulting fine and almost invisible scar with no tendency towards stretching or keloid formation; all these should commend this combined superficial and deep continuous suture as ideal for the closing of all clean wounds in abdominal surgery.



DIRECT INGUINAL HERNIA INCIDENT TO INDIRECT HERNIA: A METHOD OF DETECTION AND REPAIR*

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INTRODUCTION

THE purpose of this article is to show a method of repairing and detecting a direct hernia during the process of an indirect inguinal herniotomy. It is a well-known fact that many patients return to the surgeon, after an indirect inguinal herniotomy with recurrent hernia. Statistics prove that some of these are not recurrences, but direct hernias that had been overlooked during the first operation. This may have been due to the presence of a small sac which could not be found by ordinary means, or to a weakened floor at the apex of Hesselbach's triangle or to a displaced conjoined tendon. The unaided eye cannot see these conditions with any degree of precision. Until recently, there were no definite methods employed to detect these anomalies. Using Cameron's right-angle surgilite for illumination, as described in this paper, these defects can be readily found, and steps taken to repair them. By using the surgilite routinely there will be no excuse for overlooking a direct hernial sac, regardless of its size, a displaced conjoined tendon or a weakened floor. Surgeons, as a rule, have poor tools to work with and are slow in selecting new instruments. Nevertheless, I offer this suggestion of using the right angle surgilite as a means of eliminating the worst embarrassment that confronts a surgeon after an indirect inguinal herniotomy, that is, the so called "recurrent hernia."

The method of repairing the area involved will be described later in detail. The object is to strengthen the floor by bringing the rectus fascia and muscle underneath the conjoined tendon and

suturing them to the fascia covering the iliopectineal portion of the pubic bone. The conjoined tendon is then sutured to the rectus fascia and to the shelving portion of the inguinal ligament. This will strengthen the floor, thereby preventing any possibility of direct hernia, and also preventing femoral hernias.

SKIN INCISION

The incision for an inguinal hernia should be made long enough to permit wide exposure of the entire inguinal canal. For this reason it should extend from the pubic tubercle to at least 1 inch above the mid-point between the anterior spine and the pubic tubercle. The line of direction is made 1 inch medial to this point, and carried diagonally downward to the pubic tubercle (Fig. 1).

The above incision will suffice for indirect hernia, but if a direct hernia is present, another incision is made parallel to the pubes. This extra incision allows greater exposure for dissection at the apex of Hesselbach's triangle.

EXPOSURE OF POUPART'S LIGAMENT

Poupart's or the inguinal ligament is the thickened lower border of the fascia of the external oblique folded upon itself. It is attached to the anterior superior spine, to the pubic tubercle and the lacunar ligament. When the incision through the skin and superficial fascia has been made, it will expose the fascia of the external oblique. If a large sac is present the external abdominal ring will be seen quite readily. A groove director is inserted at the medial border of the ring and extended upwards in the direction of the

* Submitted for publication November 2, 1928.

fibers of the external oblique. These are split with the director acting as a guide, being careful that the ilio-hypogastric

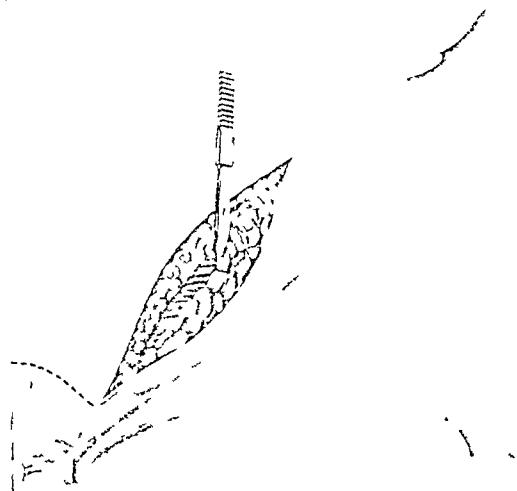


FIG. 1. Skin incision. Dotted line shows direction of extra incision in case direct hernia is found.

nerve is not within the director. With gauze dissection Poupart's ligament is freed on its anterior and posterior surfaces, thus exposing its shelving portion from the internal ring to the pubic tubercle. The fascia of the external oblique is not disturbed unless the floor at the apex of Hesselbach's triangle has to be repaired.

MOBILIZATION OF THE SAC AND CORD

When the fascia of the external oblique is opened the cord and sac should be found without much trouble. But as often occurs, the sac may be too small and will defy even the best surgeon to locate it. It may be found at the region of the internal ring. Both the sac and the cord are lifted from the floor of the canal. Dr. Carl Beck suggested the use of the right angle surgilite to distinguish the cord from the sac, thus preventing the possibility of injuring the cord. The surgilite is placed against the sac and cord, and with a knife or scissors, they are easily separated with minimum trauma. When the sac has been freed down below its neck, it is opened and its contents reduced.

USE OF THE SURGILITE

When the contents of the sac have been thoroughly reduced the surgilite is inserted

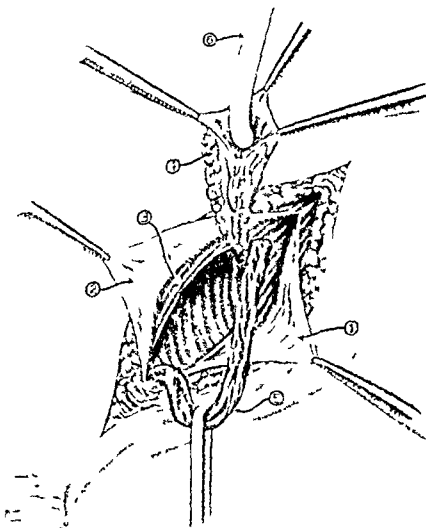


FIG. 2. Sac (4) has been separated from cord (5). Surgilite (6) ready to enter abdomen through opened sac. 1. Inguinal ligament. 2. Fascia of external oblique. 3. Conjoined tendon.

into the sac and directed into the abdominal cavity towards the pubic tubercle. Figure 2 demonstrates the method of approach. Figure 3 shows the surgilite up against the tubercle, transilluminating the thin layer of fascia transversalis. Figure 4 shows the end of the surgilite transilluminating a direct hernial sac. In these cases where the sac is present the surgilite will slip into it without difficulty. If a sac is not present and the light is freely transilluminated, it means that the floor is weak, or it may show a displaced conjoined tendon. It is known that direct hernias may be due to a conjoined tendon that inserts towards the symphysis rather than the tubercle. In such a case the floor is weakened, inviting herniation. All these conditions can be transilluminated, and by the difference in the density of the shadow cast the strength of this area can be determined. A diffusely transilluminated field means that it is covered by a thin layer of fascia; whereas, if the light is not freely transilluminated the floor is of good strength.

The amount of resistance which the surgilite encounters against the tissues will also help to determine the strength of the

tional incision is made parallel to the pubes for a distance of about 1 in. from the tubercle. The superficial fascia is also

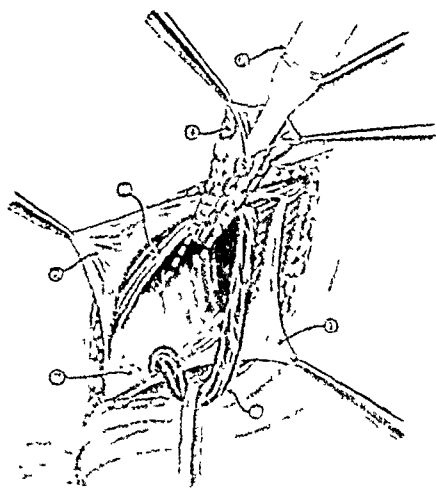


FIG. 3. Tip (7) of surgilite transilluminating apex of Hesselbach's triangle. 1. Inguinal ligament. 2. Fascia of external oblique. 3. Conjoined tendon. 4. Sac. 5. Cord. 6. Surgilite.

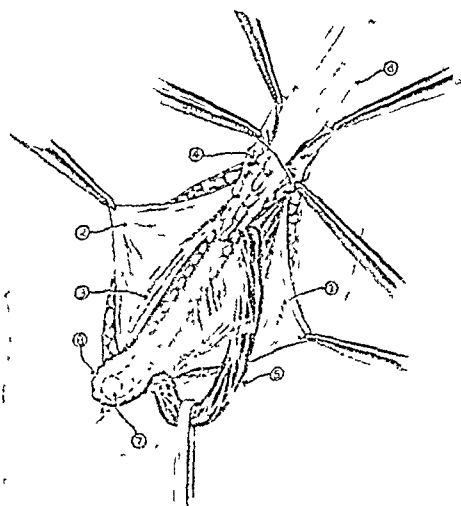


FIG. 4. Tip (7) of surgilite transilluminating direct hernia (8).

wall. I am positive that if the surgilite is used routinely in all cases of indirect inguinal herniotomy, it will be of great value to both the surgeon and the patient.

cleaned for the same distance, exposing the fascia of the rectus muscle. An incision now is made at the lateral border of the rectus, beginning from the symphysis and carried superiorly for a distance of 1 in. The conjoined tendon is dissected about

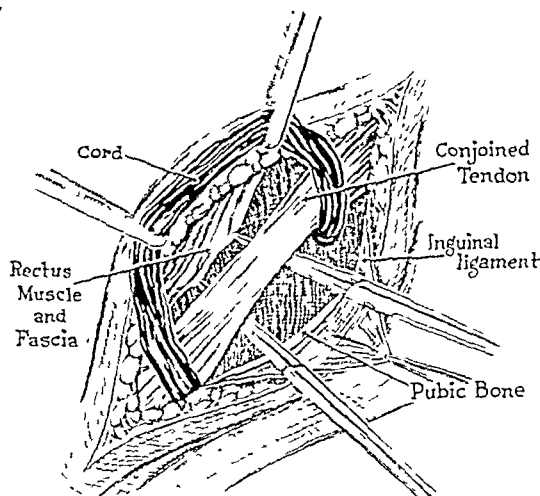


FIG. 5. Diagrammatic sketch illustrating rectus fascia and muscle picked up by two forceps underneath conjoined tendon.

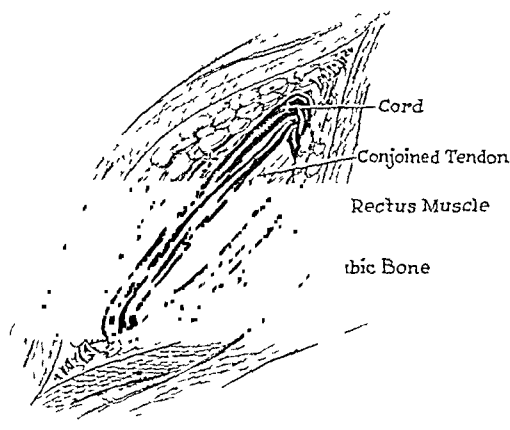


FIG. 6. Rectus fascia and muscle sutured to fascia surrounding iliopectineal portion of pubic bone. Medial border of conjoined tendon sutured to anterior surface of rectus.

REPAIR OF A DIRECT HERNIA

If a direct hernia has been found, or if the apex of the triangle is weak, an addi-

the same distance and lifted up. With forceps the rectus muscle and fascia are picked up and pulled underneath the

conjoined tendon (Fig. 5). The iliopectineal portion of the pubic bone is exposed for a short distance from the tubercle and the rectus fascia and muscle are sutured to this with two or three interrupted stitches (Fig. 6). When inserting these stitches care must be taken not to injure the femoral vein.

The medial border of the conjoined tendon is sutured to the anterior surface of the rectus fascia. The operation is completed as in any standard method, preferably the Andrew's imbrication method. The conjoined tendon with the fascia of the external oblique is sutured to the shelving portion of Poupart's ligament underneath the cord. The latter is covered over by the fascial flap of the inguinal ligament.

Following the above procedure the floor at the region of Hesselbach's triangle will consist from within outward of the following tissues: peritoneum, rectus muscle and fascia, conjoined tendon, fascia of the external oblique, the cord, and anterior to it the inguinal ligament.

CONCLUSION

1. Cameron's right angle surgilite is of great value for lighting purposes and helps to separate the sac from the cord.
2. The surgilite, when used routinely, will eliminate secondary operations and embarrassment to the surgeon.
3. The method of repairing a direct hernia is ideal in that a good strong wall is formed.
4. The method can also be used to repair femoral hernias.



ELECTROSURGICAL CIRCUMCISION*

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GENERAL FEATURES

CIRCUMCISION as the term is usually employed consists in the removal of a portion of the prepuce for the purpose of allowing the exposure of the glans.

remembered that the frenum is to be avoided as cutting at this point gives much postoperative discomfort.

The instruments necessary in this operation are:

1. Four-pronged special dilator.



FIG. 1. Special dilator, open. Showing four prongs which stretch the prepuce.

The amount of foreskin to be removed should be calculated so that two objects be accomplished. First, that enough prepuce be removed to insure easy exposure of the glans penis even when the organ is at its maximum during erection. Second, that enough foreskin be left to cover the glans during relaxation of the organ preventing drying and a loss of sensation of the organ. An exposed glans penis is a discomfort to its owner and a discredit to the operator.

The chief indication for circumcision is phimosis which may be congenital, pathological or traumatic. It may be indicated in order to reach diseases of the glans. In the manipulations during circumcision, one must remember that the skin and mucous membrane of the prepuce move freely upon the connective tissue bed common to both and unless special care be exercised in adjusting the dilating instrument, the mucous membrane will be longer than the skin. It is also to be

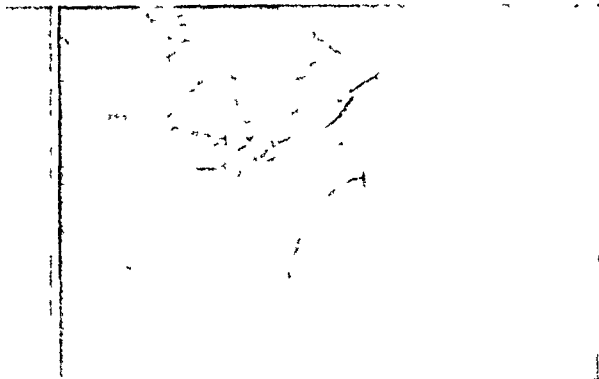


FIG. 2 Same as Fig. 1. Closed.

2. Electrosurgical apparatus.
3. Skin needle.
4. Ochsner's clamp.

1. The dilating instrument consists of four prongs $3\frac{1}{2}$ in. long, adjusted by a screw at the base, allowing the blades to be completely approximated when closed. When open, it has a circumference of 6 in. The blades are turned up at the ends and sharp in order to approximate and hold the skin and mucous membrane together. (Fig. 1.)

2. Electrosurgical apparatus producing an efficient cutting current. This current cuts as quickly as the scalpel, diminishing bleeding and, by closing small capillaries, blood vessels and lymphatics, preventing infection. The apparatus may be of the spark-gap type, producing a damped current (unequal oscillations) or a radio-tube current machine giving an undamped current (equal oscillations).

3. Needle, straight skin needle (See Fig. 3).

4. Small Ochsner clamp. (See Fig. 4.)

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Preparation. Routine preparation as in any other operation. The foreskin is retracted, cleaned with soap, water, ether

snugly about the glans. The foreskin is then pulled forward until the point is reached on the skin where amputation is

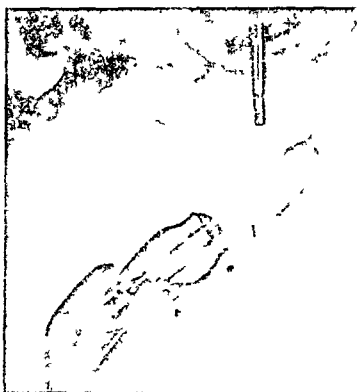


FIG. 3. Dilater has been inserted and skin incision begun.



FIG. 4. Skin and m.m. have been removed and vessels are being coagulated by Ward's technique of applying electrode to clamp.

and alcohol. If unable to retract foreskin, wait until the patient or part is anesthetized and insert dilating instrument which exposes the glans for cleansing. After the parts have been cleansed, the patient is draped as usual for surgical operations, and a sterile rubber sheet is applied.

Anesthesia. Ether, local or nitrous oxide, depending on preference of operator.

Position. Recumbent on a narrow table. The table is insulated by means of rubber mats or tires. See that no part of the patient's body comes in contact with the metal part of the table.

Advantages. In my series of 90 cases, I found this method of circumcision, to have several advantages over the older cutting methods, namely:

1. Simple technique.
2. Sealing blood vessels and lymphatics.
3. Heals first intention.
4. Little postoperative discomfort.

Technique of Operation. Holding the penis in the left hand, the closed dilating clamp is inserted in the outer margin of the prepuce. The dilator is opened and if there are adhesions between the glans and foreskin they are loosened and the field cleansed with soap, water and bichloride. The dilator is then removed and inserted so the tip of the prongs are just anterior to the corona, the four prongs fitting

desired. The prepuce is dilated until it forms a tight band around the dilator which is pulled forward beyond the end of the organ. The cutting current is applied just proximal to the tip of the prongs of the dilator. A complete circle is made



FIG. 5. Dilater reapplied to approximate skin and m.m. and stitches inserted.

with the cutting current, separating the skin only which retracts. A second circle is made with the cutting current severing the mucous membrane at the border of the retracted skin. Bleeding points, if any, are clamped and touched with the needle causing coagulation of the vessels and a cessation of bleeding. The dilator is reinserted, the skin and mucous membranes

grasped equally and dilated. An ordinary skin needle armed with No. 1 plain catgut is passed through the skin and mucous membrane at four points in the circumference dividing it into four equal quadrants. These sutures are tied and ends left long to tie in the dressing as usual.

The dilator is then removed and a small piece of vaseline gauze tied in place with the above sutures which have been left long over the raw surface and allowed to remain until the sutures are absorbed, when the wound is always healed.

By using this method, the smaller blood vessels and lymphatics are sealed when

cut. The larger vessels are grasped with an Ochsner forceps and touched with the current used in cutting. This coagulates the blood and seals the vessel. In clamping the vessel, as small amount of tissue is grasped as possible limiting the amount of tissue to be absorbed. The depth of destruction of the tissue is about $\frac{4}{10}$ mm. allowing healing by first intention. The feature of this operation that appeals to the operator and patient alike is the small amount of postoperative pain. This is due to the destruction of the nerve endings at the site of operation which are particularly sensitive.



GENERAL PRINCIPLES IN THE TREATMENT OF FRACTURES FROM THE STANDPOINT OF THE PHYSICO-THERAPIST*

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ONE can safely say that there never occurred two fractures, or for that matter two pathological conditions, that were exactly alike. They differ in location, in the form of fracture, in the age and constitution of the injured, in the condition of the surrounding tissue, in the amount of haemorrhage, in the complications—and last but not least in the time passing between injury and first aid and the subsequent treatment.

Still, in view of the fact that the injured is as a rule a healthy individual and healthy individuals react in a fairly analogous way against a traumatic injury, we are entitled to establish general principles in the treatment of fractures which may guide though not govern our actions. But routine here as everywhere else very often may lead to disastrous results.

Following the course of events we shall consider what may be the best methods of procedure from immediately after the trauma until the complete recovery or definite result. We shall therefore discuss the first aid, the setting of the fracture, the immobilization, massage, passive and active motion, use of heat in its various forms, mechanotherapy, treatment of the muscles, non-union—all this, of course, from the standpoint of the physico-therapist.

FIRST AID

In my experience patients have often reported that they had on their own account or on the advice of their physicians used hot applications immediately after an injury. Such a procedure as a routine is hardly defensible on physiological grounds. Injuries of even medium intensity are always accompanied by injuries

to the vessels at the respective location. They are always followed by hemorrhages into the surrounding tissues. Heat in any form brings about a dilatation of the peripheral vessels and hyperemia of the skin which however would be impossible without a concomittant dilatation of the deep vessels. Heat will thus cause an increase of hemorrhage and blood extravasation. While it may not often cause serious trouble it may sometimes have bad consequences, e.g. when the hemorrhage has taken place inside of a strong fascia, as the one on the forearm, and may increase the danger of ischemia.

The proper treatment would be cold applications, which do not always mean ice bags as these may give rise to severe pain, probably caused by the spasm produced by the intense cold. A compress kept cold by frequent changes or the application of small pieces of ice will give prompt relief. There may, however, occur cases where the spasm caused by the trauma is so strong that its alleviation is imperative; in these cases heat applications may be in order. The probability of an increased blood extravasation has to be accepted in such cases as the lesser evil.

Unless an expert is at hand no effort should be made to set the fracture but the injured limb should be placed on a splint and *loosely* tied.

SETTING THE FRACTURE

Strictly speaking, the setting of fractures does not come within the realm of physical therapy. Still, it has happened frequently that an unsatisfactory result has been attributed to the person who gave the after-treatment, though the blame should

* Submitted for publication November 20, 1928.

have been taken by the surgeon who set the fracture. It is for this reason that I enter into this question and the object of this section is not to give the technique of the procedures but to point out general rules which are of importance for the after-treatment.

Under the present ruling of the courts a physician is obliged to have a picture taken of every injury in which there is a question of fracture. This rule, has however, harmed the physician. We have in fact nearly lost the ability of making a clinical diagnosis of a fracture.

When the existence of a fracture has been established it is necessary to determine whether an effort should be made to set the fracture, i.e. to try to bring the fragments into a perfect apposition. This question can by no means always be answered in the affirmative.

The aim of the treatment of a fracture is in the main threefold: good union, good function and good cosmetic effect.

It goes without saying that good union is of the greatest importance as the two other factors are often dependent upon it. Good union is in general possible only if the fragments are in apposition or connected by periosteum.

Very often, however, a perfect alinement is impossible to obtain, either because one fragment is too small or because the fracture is comminuted. In spite of this a good result can be expected with reasonable certainty without any further efforts on the part of the surgeon, and the question arises whether it would be of value to try to interfere by setting the fracture. It is even possible that circumstances may arise which will relegate the injury of the bone to second place.

Sometime ago I was called to see a young lady who had injured her shoulder. The roentgen rays showed a fracture of the external tip of the right clavicle. The fragment was about half an inch long, comminuted and slightly displaced upwards, but in fair apposition. The physician who had been called immediately

after the accident tried to press the small fragment down and to hold it there with a strip of adhesive plaster which he placed over the fragment and under the elbow. This adhesive plaster bandage caused excruciating pain in the fracture and, also, caused a pressure sore on the elbow. It is not necessary to say that the effort of the physician was unsuccessful. The fragment did not move. I cut off the bandage and by plain massage and passive motion full function was restored within two weeks. The pressure sore took four weeks to heal. The cosmetic result was very good and the slight deformity present was easy to camouflage by a shoulder strap.

I have had occasion to see many fractures, particularly of the wrist which were impacted and in which an effort has been made to set the bone and bring about a perfect alinement. It is to state that according to roentgen ray pictures taken before and after setting the position of the fragments was exactly the same after the effort as it was before the manipulation. All the surgeon had done was to increase the traumatism and thus increase the difficulties for the after-treatment. This will happen very frequently when these manipulations are made on the strength of the roentgenogram without determining, as in the old-fashioned way, whether the fragments are movable and whether crepitation can be felt.

In many instances the patient would have been much better off if the surgeon had refrained from any interference and had started the active treatment at once.

Except in cases of emergency, no fracture should be set without an anesthetic, not so much for the sake of the patient and save him pain as to obtain a perfect relaxation of the muscles and to be able to manipulate the fracture without adding to the injury of the tissues. Many surgeons disregard this rule, sometimes from sheer bravado, sometimes from sheer brutality. The results will hardly ever be perfect. In former years when general anesthesia was connected with some risk such a

procedure might have been excusable. Today this practice should be condemned.

Next to good union comes good function. There are, however, many physicians who are so absorbed in the endeavor to obtain good union that they forget the function altogether. If only the fragments knit, no matter where the fracture is located, they hardly think of the function, as if this came by itself. Of course, the union of the fragments must be considered first as of greatest importance and it depends on the type of fracture how soon one must take care of the joints which are determining in the function. The nearer the fracture is to the joint, the quicker the after-treatment must be started.

For the treatment of the various fractures special attention must be paid to the particular needs in the individual cases. To illustrate this problem I wish to report a case which I saw a few years ago. I had a nurse who had been working in my office for years and had become an efficient masseuse. Her mother, a woman of about sixty had fallen and injured her hand. The hand swelled and no deformity was noticeable. Having treated similar cases in the office she starts to treat her mother at once with massage without consulting a physician. After three weeks the patient had perfect function but a marked bayonet deformity became apparent. There was nothing to do as the union was complete and in view of the perfect function a refracture would not have been indicated. It goes without saying that a perfect function could also have been obtained if the fracture had been set properly and treated in the correct way. But I believe that the patient who lived in the country would most likely have been left with a bad wrist joint if she had been treated by a doctor who was not familiar with the modern care of fractures and who put the fracture into a plastercast after the setting.

This leads us to the question of good cosmetic result. This depends in the first place on the sex. A disfiguring injury can

change the whole life of a woman while it may be of only small importance to a man. A deformity of a forearm with perfect function will be trivial for a man whose arm is usually covered. The same injury will mar a woman forever and will during the present fashion trend be a continuous embarrassment. The same holds true of a fracture of the clavicle.

Of great importance also is the occupation and the social position of the patient. A woman of the working class, particularly if she is married, will be better served by a good function of the limb than by perfect cosmetic effect, while a man whose social position is high does not like to be conspicuous because of disfigurements.

The age has likewise to be considered. An older woman as well as an old man will be able to adjust her or himself easier and quicker to a deforming injury than a young person.

Thus, in setting a fracture the physician should consider these three points: union, function and cosmetic effect. He may refrain from all action if the outcome is doubtful; he may sacrifice for the sake of the most important issue one of the other factors; and he may in another case where the pathology is analogous act in an opposite manner. *All this of course if he cannot fulfil all three indications.*

In some cases the patient constantly asks whether the fracture was properly set. The physician however should have the courage of his conviction and realize that in some cases an improvement in the apposition is impossible and that any energetic effort to loosen the fragments will only aggravate the traumatism, thus disturbing the regenerative processes, and interfering with the reestablishment of function, and all without obtaining the object desired.

It may be in order to state that the foregoing paragraphs do not insinuate that one should not try to obtain a perfect result from the point of union, function and cosmetics. One must however weigh the indications and possibilities and not, in trying to get the impossible, let the

opportunity slip to obtain a result which was within one's grasp.

IMMOBILIZATION

It may sound obvious but it should be expressly stated that immobilization is not a treatment of fracture per se. Immobilization has various objects. It is used to hold the fragments in apposition after the fracture is set, but it may also be used to allow the injured part to relax by preventing movements which would cause pain.

Therefore when we immobilize an injured limb we ought to keep steadily in mind the purpose of the immobilization. If the fragments are movable we must immobilize until a certain amount of fibrous union has taken place. It will then depend on the form of fracture and on its location how long this should be continued.

Experience has shown that oblique and spiral fractures are more likely to slip and get into a bad position again than are transverse ones. A fracture in the middle of a long bone will not stay as well in the position into which it is placed as will a fracture near the joint. To this rule, of course, there may be and are exceptions.

As soon as a fibrous union has developed the purpose of the immobilization has changed. It is used to protect the fibrous union which already exists.

This analysis is by no means a sophism or hair-splitting. As long as no union exists the immobilization must not be interfered with lest the apposition be disturbed. Early active treatment should not be tried as the advantage gained would not be worth the chance taken. If a fibrous union has developed we are sure of a certain amount of coherence and can institute active treatment if we exercise proper care. The fragments are held together and we use the immobilization as a protection against a new, though probably slight, injury which could break up the fibrous union.

If, on the other hand, the fragments are

impacted or held in place by other means, or if one fragment is so small that it can be neglected, immobilization can have one object only, to prevent or stop pain. It is then obvious that active treatment may be started at once as the fragments are united anyhow or should even be prevented from uniting. The indication to be fulfilled in these cases is therefore not immobilization of the fragments but the relief of pain and reduction of the swelling. The immobilization is not the main goal, it is only a special modality.

Here again it shows that we should not ask ourselves what we should do in a certain case but what we want to accomplish, not which form of treatment should be used but what we expect from the procedure. Once the problem is properly stated the solution will be easier. At any rate the planless experimentation, the pure empiricism and the glorification of precedence will be restricted to cases where our knowledge is entirely inadequate.

Once the true part immobilization plays in the treatment of fractures is recognized, routine treatments becomes impossible. We will choose plaster casts, splints, etc. according to the peculiarities of the case and not on account of the location of the fracture. Surgeons often take off a cast and replace it with an equally heavy one because a callus is still soft, though the bone is not to be actively used, for example, in Colles' fracture. A light removable starch bandage or a light splint would serve the purpose just as well, the purpose being protection against an injury.

Wherever circumstances permit immobilization should be done in such a way that immediate treatment of the injured part is possible. This is shown in the treatment of fractures of the upper part of the humerus, where a form of immobilization will be possible which holds the fragments very well and safely, and still the shoulder may be kept uncovered so that active treatment may be started. Such arrangement can be made in various fractures.

In a case of Colles' fracture with marked

swelling of the forearm and fingers the cast, if one is necessary, should under no circumstances prevent the motion of the fingers. In most of these cases the edema and stiffness of the fingers persist long after the fracture is healed.

MASSAGE

Massage should be started as soon as possible, depending on the kind of fracture; in impacted fractures where a danger of displacement of the fragments does not exist it may be begun at once, in other fractures as soon as this danger has disappeared. Fanatism in this work is to be condemned. We should treat the patient according to the needs of the case and not according to principles. Thus, fracture of the femur shafts should be treated late, rather too late than too early. No greater calamity is possible than a faulty union in the femur or the tibia. I fully appreciate the advantages of early massage in all fractures, but there are those in which taking the slightest chance would be foolhardy.

The massage must be given with the greatest gentleness; only stroking can be used, with the hand of the operator fully relaxed. Only the fully relaxed hand will bring about the full relaxation of the spastic muscles in the first days after the injury. Only the fully relaxed hand can adapt itself perfectly to the contour of the affected part. The motion is done from the shoulder; the hand forms merely an attachment. Masseurs who move their fingers against one another do not understand the technique used in fracture treatment.

It seems to me that the rule made by competent authors that the place of the break should not be massaged early, that the hand should avoid these parts while massaging, is an unwarranted generalization. There are cases which show a very marked *hyperesthesia* over the injured part. To manage these parts is very disagreeable to the patient. However, I can not conceive of a physiological reason to adhere to this rule in general.

The object of the massage is what I term an internal *debridement*. The wreckage left by the injury and the hemorrhage should be carried off as soon as possible in order to minimize the regenerative inflammatory reaction with all the dangers following in its wake. The sooner this is done the more favorable is the situation for quick removal. Physical therapy, particularly massage, can do it. Massage in the later stage, when the swelling and the blood extravasation have disappeared, is of very little value. Its effect on the muscles *atrophied* by disuse is far better replaced by stimulation with the electric current.

MECHANOTHERAPY

Passive and active motion must be started as soon as possible, that is as soon as they can be given without causing pain.

Due to a widespread misconception of the action of mechanotherapy physicians believe mobilization must hurt. Mobilization should not be allowed to hurt for the following reason: If we move a joint and cause severe pain, the patient goes away with a sore joint and he will not move it but keep it immobilized; thus all the value of the treatment is lost. However, if the patient leaves with a joint less painful than before, he will move for the sheer pleasure of seeing the motion. Any violence in the treatment defeats the very object we are working for, the active motion.

For the purpose of reducing the pain during the mobilization we use the various forms of heat.

Though many physicians advocate the use of diathermy a few days after the injury, it seems that hot air or infrared rays are more advisable, as technically diathermy cannot be applied efficiently. In the early stages of a fracture the swelling of the injured part is often too extended and a general heat application fills the indication to improve the circulation much better. Hot whirlpool baths and motion under the influence of the infrared rays

are easily executed on account of the decreased spasms in the muscles.

In cases where no swelling is present or where a stiffness and pain in the joint persists after long immobilization diathermy is the only efficient treatment. The plates should be placed in such a way that motions, passive as well as active, can be performed while the current is on. The passing of the diathermy current relaxes the tissues around the joint, reduces the tenderness and thus under the treatment, excursions of 10 to 30 degrees beyond those before the treatment can be executed without pain.

The passive motions are, if possible, done by the patient, inasmuch as he or she supplies the force necessary to flex the joint. Thus, if the right elbow is to be bent the force to bend may be supplied by the left arm. In this way we are assured that the patient will go as far as he can, that he will not resist but assist the motion. One can see over and over again that a patient making satisfactory progress is thrown back days and weeks by brutal attempts at mobilization. If a patient is improving there is no need to try beyond the range of active motion.

TREATMENT OF THE MUSCLES

That the spasm of muscles is relieved by gentle massage immediately after the injury has already been mentioned.

As far as the prevention of atrophy of the muscles is concerned massage has only a limited value. Massage only aids the circulation passively. It increases the arterial circulation only by hastening the venous flow. The method which approaches the normal contraction is the stimulation

with electrical currents. Through the stimulation an active functional hyperemia is produced which is helpful in strengthening the muscles. Which type of apparatus to use is of secondary importance, as long as a contraction is produced. The most pleasant form is the tonisator and the sinusoidal-faradic.

NON-UNION

In dealing with this serious condition various forms of treatment have been suggested, e.g. passive hyperemia, ionization, diathermy. The effectiveness of these procedures is very difficult to prove. Besides, non-union is due to various causes and it is obvious that a treatment which does not fulfill the indications set by a particular case can have no value at all.

Diathermy will be of value where the nutrient arteries of the bone are destroyed and a new circulation must be established.

Ionization will stimulate the activities of the tissue and passive hyperemia may or may not favor the development of connective tissue and the calcification.

In laying down these principles it is in no way the intention of the writer to dictate to the surgeon what to do in his particular sphere.

The man who gives or is in charge of the after-treatment is the one who realizes what should have been done to prevent unpleasant, unfavorable conditions. The surgeon who refers his fracture cases for physical therapy is not always aware of the fact that the slow progress in the cure is due to the disregard of fundamental principles on his part and not to the inefficiency and worthlessness of physical therapeutics.



A NEW STATISTICAL STUDY OF CANCER*

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ALL statistical reports of deaths are the compilations of punch-card records which necessarily tabulate malignant deaths in groups and omit much of the information which appears upon death certificates. For the purpose of giving publicity to certain interesting data, to give surgeons a knowledge of the relative appearance of certain tumors and the frequency of certain cancerous involvements, and to determine some improvements needed in making official records, this study was undertaken. It involved an inspection of every original death certificate filed during one year in one state and a tabulation of twenty items from each certificate of death from cancer, most of which are never considered in the usual statistical compilations.

In Pennsylvania in 1927 there were recorded exactly 120,000 deaths; the deaths exclusive of stillbirths numbered 111,273. Malignant tumors caused 9276 deaths, 8.3 per cent of all deaths, giving a general death rate of 95.4 per hundred thousand population. Among these malignant tumors the deaths reported as due to carcinoma or cancer numbered 8673, with a death rate of 89.1. Adding to these the 471 sarcoma deaths gives a total of 9144, with a death rate of 93.8. The balance of 132 deaths are from other tumors classified statistically with the cancers. The main groups of the International Classification had the following deaths from cancer and other malignant tumors; buccal cavity 300, stomach and liver 3390, peritoneum and intestines 1349, female genital organs 1347, breast 852, skin 227 and other or unspecified organs 1811. These seven groupings are necessary in ordinary statistical work as it is impossible to list in tabulation the countless separate locations where cancer may occur, nor is it possible ordinarily to list separately

the various kinds of malignant tumors. It is therefore impossible for anyone from regular mortality tables to make a study of carcinoma of a separate organ, as the tongue or thyroid. One of the purposes of this study was to collect data of individual organs and separate the various kinds of malignant tumors as accurately as was possible according to the statements of the attending physicians. A similar study has not heretofore been attempted.

The important locations of involvement among 8673 deaths designated as due to cancer or carcinoma, with the number of deaths from each, included the following: stomach 2104, intestinal tract 1740, female generative organs 1347, breast 818, liver and gall bladder 744, pancreas 334, bladder 291, prostate 245, esophagus 132, lungs 91, larynx 70, tongue 68, thyroid 29, mediastinum 19, tonsil 18, kidneys 19, penis 19, testicles 16, parotid 11, submaxillary gland 5, adrenal 3, scrotum 3, spinal cord 2, seminal vesicles and epiglottis each 1, and 1 carcinoma of the pituitary gland discovered at autopsy. The following were reported as sarcomas: kidney 21, lungs 18, testicles 10, eyes 7, prostate 6, spleen 5, bladder 4, thyroid 3, penis and parotid each 2. The miscellaneous malignant tumors not included in the above figures caused 1811 deaths and included such tumors as osteosarcoma 108 deaths, brain tumor 70, lymphosarcoma 50, hypernephroma 49, endothelioma 9, teratoma 2, and over a dozen other kinds of tumors of lesser importance.

A questionnaire was sent to 326 hospitals of the state and data were received or collected from 239 hospitals which courteously cooperated in the work. The varying systems of keeping hospital records prevented complete replies from all.

Seventy-four hospitals reported the

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number of their cancer deaths occurring during 1927. The death certificates of these same hospitals showed them to have had 1588 cancer deaths, or 18 per cent more than they acknowledged. Sixty-seven hospitals reported 411 fewer deaths than the death certificates showed and 50 claimed having had 107 more cancer deaths than their original certificates attested. These discrepancies damagingly vitiate the value of data collected by the questionnaire method and suggest the lack of value of such a system for formulating conclusions. They are due, in the first case, to incomplete hospital records, or to an incomplete system of keeping them, or to guess-work rather than thorough search by the clerk answering the questionnaire. In a few instances they were due to the selective system of the International Classification of the Causes of Death in giving preference to cancer over various diseases in determining the statistical cause of death; for instance, a man with intestinal carcinoma may be regarded by the hospital authorities to have died of appendicitis or intestinal obstruction and to be so classed, whereas statistical procedure would class the death as due to cancer, even if fatal forms of appendicitis, diabetes or tuberculosis were present. Hospital records will show more cancer deaths than death certificate compilations when the death certificates do not give the hospital as the place of death. The name of the hospital or other institution where death occurred is frequently omitted; this, with the home address of the patient, should always be written on the death certificate.

CANCER OF THE BREAST

Cancer of the breast caused 818 deaths, or 8.8 per cent of the deaths from all malignant tumors, or 9.4 per cent of the deaths from carcinoma. The death rate from this disease increased from 1.7 in 1910 to 2.1 in 1920, an increase of 2.3 per cent in these deaths per year. The prevalence of cancer of the breast was suggested by the replies from 237 hospitals which had 1029 cases with 7.3 per

cent deaths in the hospital. On the death certificates giving information about operation it was found that 54 per cent of the women were operated upon with an operation mortality of about 5 per cent and that one-fifth died in the hospital. The survey showed that the number of women operated upon for cancer of the breast was twice the number of deaths from this disease; the hospitals of the state reported over 900 operations for this disease during the year in which 400 women who had been operated upon for this disease subsequently died of cancer.

The death rate from cancer of the breast among white women was 52, among colored women 26, per hundred thousand women over thirty-five years of age. The marriage state seems to have had no effect upon the production of cancer of the breast, it having caused nearly three times as many deaths among single women as among those who had been married. The death rate of cancer of the breast of single women was 109, of women who had been married 44, per 100,000 women over thirty-five years of age in 1927.

The ages of the women dying from cancer of the breast ranged from twenty-two to ninety-two, with an average of fifty-eight. The percentage of the deaths under thirty-five years of age was 4.4, under forty-five years 17.7 per cent. About one-sixth of the deaths occurred before forty-five years of age. The ages of the women with Paget's disease were between fifty-three and ninety.

The side of involvement was recorded on some certificates, right 146, left 175, both breasts 42.

Cancer of the breast in the male caused 2 deaths.

Paget's disease caused 6 deaths, one being in a male, which was in addition to the two other male cases.

CANCER OF THE LIP

Cancer of the lip caused 43 deaths, all except 4 being in men, averaging sixty-seven years of age. On the certificates designating which lip was involved there

were reported 2 cancers of the upper and 16 of the lower lip. In one instance both lips were involved. The Philadelphia and Pittsburgh Hospitals had 27 cases with 7 deaths in the hospitals.

CANCER OF THE TONGUE

Cancer of the tongue caused 68 deaths. The Philadelphia hospitals treated 35 cases and had 9 deaths in the hospitals, while elsewhere in the city there were 15 other such deaths. The Pittsburgh hospitals had 6 cases with 3 deaths. Of the total cases 93 per cent were males, with ages ranging from thirty-nine to eighty-six, averaging sixty years. The duration of the disease was recorded as varying from four months to two years in non-operated cases, with the same variation of duration in other cases after operation.

CANCER OF THE LARYNX

There were 70 deaths from carcinoma of the larynx, 90 per cent being in men whose ages varied between forty-seven and eighty, averaging sixty-five years. The Philadelphia hospitals had 72 cases with 20 deaths, while 15 other such deaths occurred in the city. The duration was recorded as varying from two months to three years; 6 of 12 operative cases died within one week following the operation; others lived two or three years.

CANCER OF THE LUNGS

Cancer of the lungs caused 91 deaths exclusive of 18 other deaths recorded as from sarcoma of the lungs. On only 10 certificates was the statement made that the cancer was primary. On 19 the cancer was on the right, 13 on the left side. The sexes were equally involved. The ages of the patients varied from thirty-one to seventy-one. The durations of the disease were recorded as from three to eighteen months. Diagnosis was made by roentgen ray in only 12 cases; the disease was discovered at autopsy in 6. The occupations of the men varied greatly, from rabbi to glassworker or cigar maker; they throw no light upon any possible causative agent.

CANCER OF THE STOMACH

Malignant diseases of the stomach and

liver caused 3390 deaths, 36 per cent of all malignant deaths. This total included 1960 deaths from cancer of the stomach, besides 144 designated as from cancer of the pylorus, 744 deaths reported as cancer of the liver, gall bladder or bile ducts and 132 esophageal cancer. Sarcoma of the stomach was recorded on 10 death certificates, of the liver on 20 others, and 1 sarcoma of the esophagus was recorded.

Cancer or carcinoma of the stomach was recorded on 1960 certificates and doubtless included many pure pyloric cancers, although the pylorus was mentioned only on 144 certificates. This is a ratio of 14 to 1. The hospital replies recorded 262 stomach and 41 pyloric cancers, a case ratio of 5 to 1; the Philadelphia hospital case ratio was 9 stomach to 1 pylorus, the death ratio 13 gastric to 1 pyloric cancer, while the corresponding Pittsburgh ratios were each 9 to 1.

The tabulations of deaths from cancer of the stomach and liver as published by statisticians include deaths from all malignant tumors of the pharynx, esophagus, stomach, pylorus, liver, gall bladder and bile ducts. This group in the 1927 deaths in Pennsylvania included the following locations of carcinoma or cancer: esophagus 132, stomach exclusive of pylorus 1960, pylorus 144, liver 774, gall bladder and bile ducts 114. Sarcoma was the diagnosis given for 7 tumors of the stomach, 18 of the liver and 1 of the esophagus.

Gastric ulcer was recorded on only 22 death certificates as having been present with cancer of the stomach.

The diagnosis was recorded on 1842 death certificates as having been made clinically 342 times, by laboratory analysis 40, laboratory combined with roentgenographic diagnosis 17, and by roentgen ray alone in 182 cases. In hospitals which were equipped with roentgen-ray diagnostic apparatus an operation was performed upon 34 persons after confirming the clinical diagnosis of gastric cancer with the roentgenogram, but upon 89 other persons operations were done

without using the convenient roentgen-ray apparatus which these hospitals had and could have used to confirm the diagnosis. Operation was recorded as having been performed upon 206 gastric carcinomas, and although about one-tenth of the death certificates are of old forms not inquiring about operations, this ratio means that operation was reported as having been performed upon about one-eighth of the gastric cancer patients. Operation was followed by autopsy in only 17 recorded instances; autopsy was done alone upon 31, and largely for diagnostic purposes.

There were only 138 cases giving the date of operation for cancer of the stomach. Twenty-two died within two days; over one-third within one week; 18 per cent lived one month, an equal proportion survived three to five months; only 4 persons lived over one year and 4 more over two years.

The average age at which death occurred from cancer of the stomach was 63.7 years. The ages at death ranged from nineteen to ninety-four. The age group sixty to sixty-nine was the largest with 643 cases, or 33 per cent of the total. The age group fifty to fifty-nine had 397 cases; the group seventy to seventy-nine had 493.

CANCER OF THE LIVER

Cancer or carcinoma of the liver, without the certificate naming any other organ in which the primary involvement could have been located, was recorded for 774 deaths. Upon only 2 certificates was the statement that the carcinoma was primary in the liver, both cases being discovered at autopsy. The gall bladder was designated as the seat of involvement of 114 other cases, the ampulla of Vater once. Sarcoma was the diagnosis for 20 other deaths from hepatic malignancy. The hospitals reported 225 cases of cancer of the liver to the question asking for the number of primary cancers of this organ. The hospitals stated they had had 107 deaths from this cancer.

The diagnosis of cancer of the liver was made clinically 139 times, by roentgen ray

12, at operation 66 and at autopsy 11. Eight operations followed a clinical and only one roentgenographic diagnosis.

CANCER OF THE PANCREAS

There were 334 deaths from cancer of the pancreas, 47 being recorded as of the head and only one of the body of this organ. Fifty-nine per cent were women. The duration of the cancer was given as from six months to four years. Half the operated cases died within one week; one of 17 patients lived two years. The average age of the operated patients with an acceptable diagnosis was: male fifty-eight, female fifty-one. Clinical diagnosis was made upon 30 of the fatal cases, and by roentgen-ray 11; operation was recorded as being performed upon 55, autopsy, 27 cases. Sarcoma, with a clinical diagnosis, was on 2 certificates of pancreatic malignancy. Ninety-three hospitals reported had 55 cases with 22 deaths in the hospitals.

CANCER OF THE INTESTINES

Malignancy of the intestines, peritoneum and mesentery caused 1349 deaths. These included deaths from cancer of the small intestine without sectional designation 290, duodenum 41, jejunum 2, ileum 9, ileocecal valve 4, cecum 71, colon without definite location 118, ascending colon 16, hepatic flexure 6, transverse colon 18, splenic flexure 10, descending colon 26, sigmoid 153, rectum 391 and anus 2. There were 12 mesentery, 19 omental, 15 peritoneal and 2 retroperitoneal cancers recorded.

CANCER OF THE RECTUM

Rectal cancers caused 391 deaths, the sexes being equally involved. The Philadelphia and Pittsburgh hospitals reported treating 150 cases and had 83 deaths in the hospitals during the year. The ages of the males dying from this cancer ranged from thirty years, the females from twenty to ninety. Most of the cases were about equally distributed along the years from fifty to seventy-five. The average age was for males sixty-one, females fifty-one, years. Collectively death certificates are very inaccurate in the declarations of the

duration of the disease causing death. On those certificates on which the duration did not correspond with the dates of treatment, the durations of the rectal cancers ranged from three months to ten years; from three to five months 15 cancers, six months 19, seven to eleven months 16; between one and one and a half year's duration 52; two years 33; over two years 19 cases. The duration of life after operation in 67 reported cases was: two days or less 8, total dying within one week 19, dying within six months two-thirds; surviving one year 12, two years 4, eight years 1.

CANCER OF FEMALE GENERATIVE ORGANS

Malignant disease of the female generative organs caused 1347 deaths, 14 per cent of all malignant tumor deaths. They included deaths from cancer or carcinoma of the uterus 930, cervix 182, vagina 16, vulva 22, ovary 93 and chorioepithelioma 5; the following recorded as sarcomas: uterus 17, ovaries 10 and vagina 1.

CANCER OF THE UTERUS

Cancer of the entire uterus caused 1112 deaths, the cervix being given on 182 certificates as the specific location. This survey cannot give the true ratio between cancers of the fundus and of the cervix. The hospitals of the state reported having treated 261 cancers of the "uterus" and 349 of the cervix; the Philadelphia figures were 104 and 124, and the corresponding reports from the Pittsburgh hospitals were 69 and 106, respectively. A wide variance on the death certificates, 930 "uterus" and 182 cervix cancers, shows the general lack of care in writing specific, important, legal documents.

The marriage state apparently has no effect upon producing cancer of the uterus. There were 121 deaths from cancer of the uterus and cervix among single women and 1224 among those who had been married, giving the death rates per 100,000 women over thirty-five years of age as 103 for single women and 79 for those who had been married. The childbearing status of these women cannot be determined.

The ages of the women dying from

cancer of the uterus varied from twenty-three to ninety, averaging fifty-five years. Seven per cent of the women were below thirty-five and 24 per cent were below forty-five years of age. The so-called cancer age of women begins at about the thirty-fifth year. The age group fifty to fifty-nine had the greatest number of deaths, 251 or 27 per cent of the whole. The death rate from cancer of the uterus for white women was 39.0, for colored 24.6 per hundred thousand women over twenty-five years of age. The corresponding rates for cancer of the uterus and cervix combined were: white 81, colored 69.

CANCER OF THE PROSTATE

Cancer of the prostate caused 245 deaths, over 7 per cent of all cancer deaths of men. The Philadelphia and Pittsburgh hospitals reported having 77 cases. The corresponding deaths in these hospitals were 38, but in these cities outside the hospitals 34. The ages of the men dying of prostatic cancer varied from thirty-nine to ninety-four, averaging seventy-two years, with the majority evenly distributed between sixty and seventy-five years. The durations of the disease were recorded as from three months to six years, averaging a year and a half. In 52 operative cases 5 died within three days, 14 died between one and five months; surviving a year were 9 of whom 2 lived four years.

SUGGESTED NEEDS

1. More and earlier operations.
2. More extensive use of available diagnostic methods.
3. More biopsal or radiographic examinations.
4. Terminal diagnosis by microscopical examination.
5. Autopsy in obscure cases for diagnosis for legal purposes.
6. A realization of the need of definiteness on death certificates.
7. More complete hospital records and death certificates.
8. A statement of all the facts in the legal death records.
9. The use of only the latest forms of death certificates.

BLADDER NECK BAR*

AN HISTORICAL AND CRITICAL REVIEW

EDWIN W. HIRSCH, M.D.

CHICAGO

IN 1834 Guthrie described the case of a man who died following urinary retention, in whom the post mortem examina-

physician, Leroy d' Etiolle (1798-1860) was giving his attention to the matter of relieving prostatic obstruction by means



FIG. 1. Specimen presented by Guthrie as evidence of entity "bar at neck of bladder." Line passes above alleged bar. Note large diverticuli.

tion revealed an apparent elevation at the bladder neck, which he termed a bar. This he construed as the cause of the retention. A glance at his specimen (Fig. 1) shows what is unmistakably a case of multiple diverticuli of the urinary bladder. It is more feasible to believe that this man's urinary difficulty was due to the large diverticuli rather than to what Guthrie describes as a bar, for it is not apparent how this bar, which in the illustration shows no occlusion of the urethra, should be deemed an obstructing element. This case, therefore, cannot be diagnosed as a bar because the enormous diverticuli are sufficient to explain the urinary retention. Writers referring to this case do not speak of the diverticuli present and are therefore unfamiliar with the pathological findings.

About the same time a young French

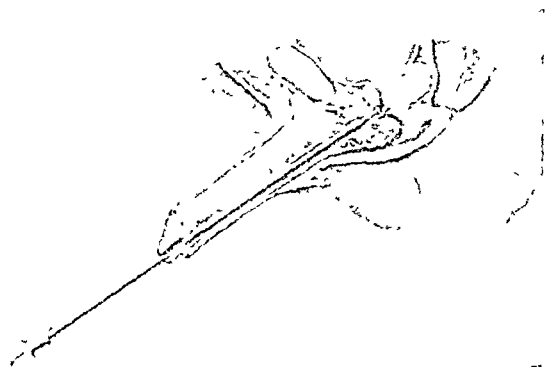


FIG. 2. Etiolle's porte-caustic. Used for burning prostate with fused silver. Bell discontinued using silver for this purpose in 1804, because of extensive strictures which resulted.

of instruments which would cut away the offending lobe of the prostate. His original instrument consisted of a lithotrite with a cutting edge which he later modified.

Etiolle designed many instruments for removing portions of the enlarged prostate, among which are to be found a caustic applicator (Fig. 2) for burning the prostatic urethra; a prostatic scarifier (Fig. 3) for scraping away a portion of the prostatic urethra; a prostatic incisor and excisor (Figs. 4 and 5) for biting away a piece of the prostate; and a prostatic ligature (Fig. 6) for snaring off the hypertrophied lobe of the prostate. It is fortunate that these instruments never found general use because they could not accomplish the purpose for which they were intended. The disadvantages attending their use were the dangers of hemorrhage and continued pain because of mutilation of the prostatic urethra. Etiolle was not familiar

* From the Department of Urology, College of Medicine, University of Illinois. Submitted for publication December 29, 1928.

with the fundamental pathology of prostatic hypertrophy; for if he were, he would not have continued in trying to remove a

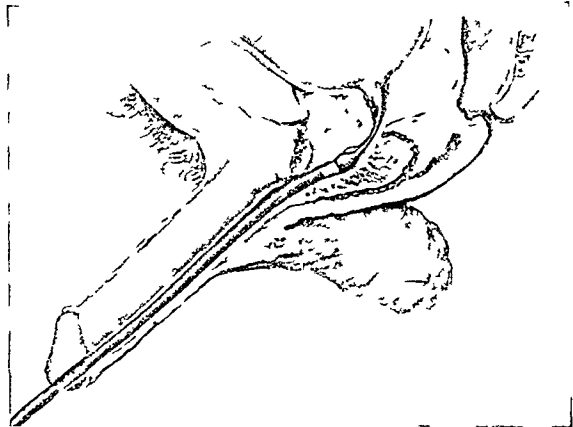


FIG 3 Etiolle's prostatic scarifier (From Bourgery and Jacobs)

growth of the prostate in a blind manner by a small cutting instrument the use of which was attended with many complications. Many of Etiolle's ideas were utilized by other urologists and incorporated as their own.

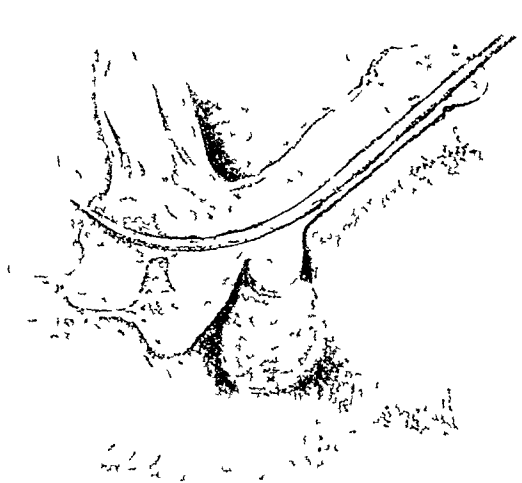


FIG 4 Etiolle's prostatic incisor, a dangerous and useless instrument. (From Bourgery and Jacobs)

Another French urologist, Civiale (1796-1867) also modified the lithotrite and advocated its use for removing obstructions at the bladder neck which he termed barriers. He was a far more profound student than Etiolle and was quite rational

and conservative in his treatment of this affection, first trying every available non-operative means.

The subject of bar formation was given added impetus by Mercier (1811-1882) who wrote many articles on the subject of valves at the neck of the bladder and claimed that his method of removing them by means of an excisor and incisor was originated by himself. On examining the case reports of his 15 selected cases, one finds that he had two deaths or a mortality of 13.3 per cent. Five cases are supposed to have had complete relief. One case was followed for four years and one for two years. Seven cases of questionable improvement were counted as cured. One case showed no improvement.

In England a more conservative method of treating urinary retention was established by Sir Henry Thompson (1820-1904) who preferred to catheterize the patient once or twice daily. As a result he had practically no mortality and afforded his patients considerable relief. Though he was

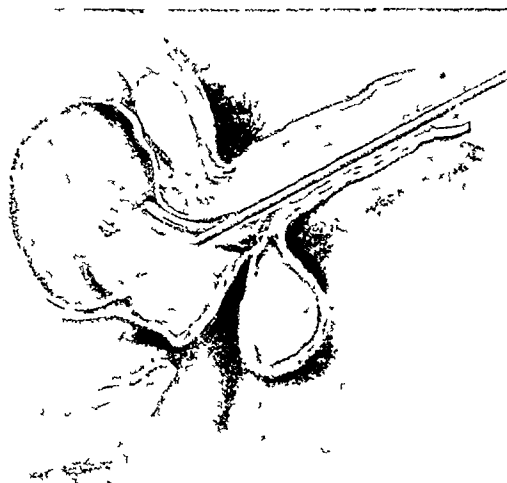


FIG 5 Etiolle's prostatic excisor. Dangers attending use of this instrument are obvious (From Bourgery and Jacobs)

aware that his method was only a palliative measure, the results were far better than the uncertain methods of blindly biting or cutting away a portion of the bladder neck. Basing his opinion on his

pathological study, Thompson stated that bars were very infrequent occurrences and consisted of an inflammatory spasm of the bladder sphincter which like the iris in similar circumstances became permanently contracted and lost the power of relaxing. Thompson considered the cause of urinary obstruction in elderly men to be usually due to prostatic enlargement.

A review of the work of Guthrie, Etiolle, Civiale, Mercier and Thompson compared to the present-day conception of bladder neck obstruction shows that Thompson had the clearest conception of the pathology of bladder neck obstruction and his humane and conservative method of treatment was a blessing to his countrymen. He would not accede to the use of trans-urethral operative treatment of prostatic bars because first, he was aware that his contemporaries were misdiagnosing bars for genuine prostatic enlargements and secondly their high mortality and uncertain results could not compare with his manner of handling such cases. Thompson prophesied that the day would arrive when the prostatic tumor would be completely removed by operative measures.

SUMMARY

Guthrie cannot be termed the original describer of a bar at the neck of the bladder because the pathological facts in the case do not indicate that his diagnosis was correct.

The benefits which Mercier claimed for his method of treatment are questionable.

Civiale was a competent urologist and made worthy contributions to urology.

Thompson is entitled to the greatest share of honor because the diagnostic and therapeutic principles which he laid down are as valid today as they were when elucidated. His work stimulated physicians to perfect methods of removing the prostate surgically because he considered the intraurethral methods of removing prostatic tissue to be unsatisfactory.

Bars are caused by a fibrosis of the bladder neck, and are not common. Many cases which are diagnosed as bar are in

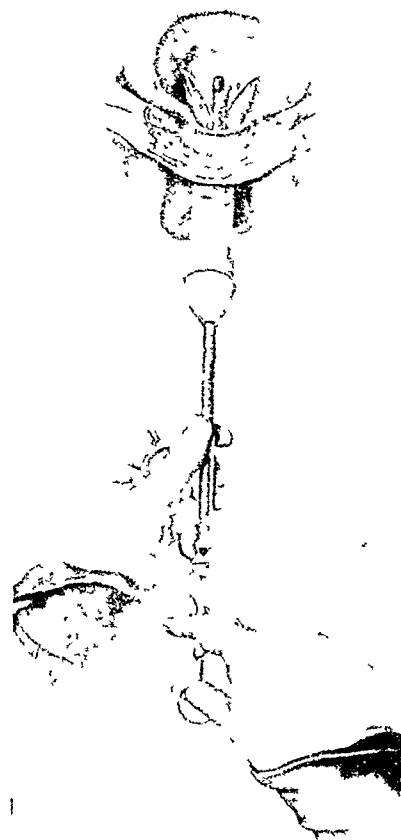


FIG. 6. Etiolle's prostatic ligature. Fanciful conception of inventor; impractical and dangerous. (From Bourguery and Jacobs.)

reality due to a small hypertrophied middle lobe of the prostate.

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/ C A S E R E P O R T S /

GIANT-CELLED TUMORS OF TENDON SHEATHS*

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TORONTO

GIANT-CELLED tumors of tendon sheaths were considered sarcomas until 1891 when Heurtaux¹ first

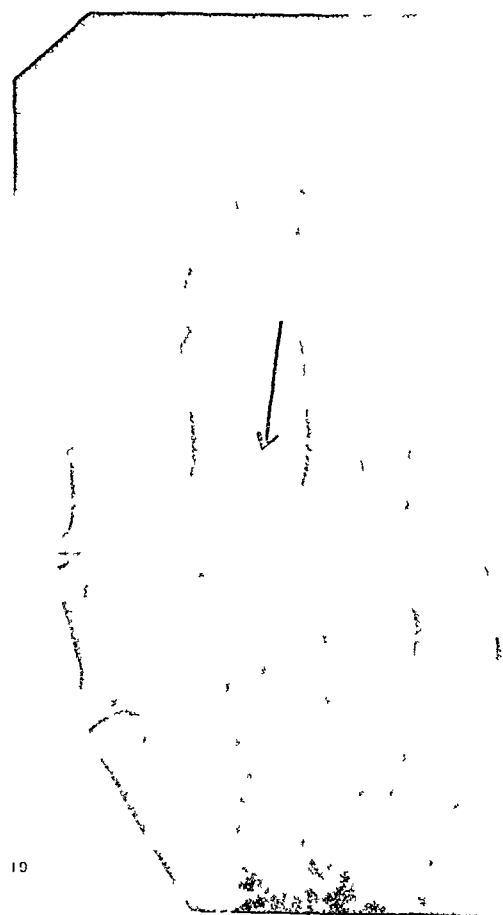


FIG 1. Radiograph taken Nov. 8, 1926, showing erosion of shaft of proximal phalanx of index finger near base.

distinguished them from true sarcomas. He called them myelomas. Targett² writing in 1897 still believed in the essentially malignant nature of these tumors. Bellamy³ in 1901, followed later by Stewart and Flint,⁴ regarded these

tumors as due to a proliferation of endothelial cells of blood vessels and hence essentially endotheliomas. At present there is still some division of opinion as to the true nature of these growths, some⁵ regarding them as true tumors while others^{6,7,8} consider them the result of an inflammatory process following the extravasation of blood as a result of trauma and hence essentially granulomas.

Gross Pathology. The tumor is usually larger the more proximal to the body it occurs. The average size in Broders' series of 17 cases was 2.8 cm. The growth is generally encapsulated and may surround tendons and nerves. It is lobulated, elastic in consistency and in color pinkish or yellowish or gray with yellowish areas due to lipoid.

Microscopic Pathology. From the capsule strips of connective tissue run toward the center carrying blood vessels which show hyaline degeneration. From these strips run bundles of fibrils surrounding nests of cells and single cells of various shapes. Giant cells are present containing a variable number of nuclei. Lipoid cells are to be seen, large and vesicular, containing round or oval-shaped nuclei often eccentrically situated. There are also to be seen pigment deposits of hematogenous origin: hemosiderin crystals.

Etiology. Injuries or infections are usually the provocative factors. These may act by decreasing local resistance to pre-existing tumor cells.

Symptoms. The commonest situation is about the metacarpophalangeal articulation usually of the right hand. The tumor grows very slowly and interference with

* Submitted for publication November 24, 1928.

function, if it occurs at all, is late. It is characteristically not adherent to skin or bone.⁹ In 1 of Broders' cases, however,

CASE REPORT

I. E., a clothing operator, aged thirty-four, was referred to me on November 8, 1926. On



FIG. 2. Low-power photomicrograph of tumor, showing large number of giant cells.

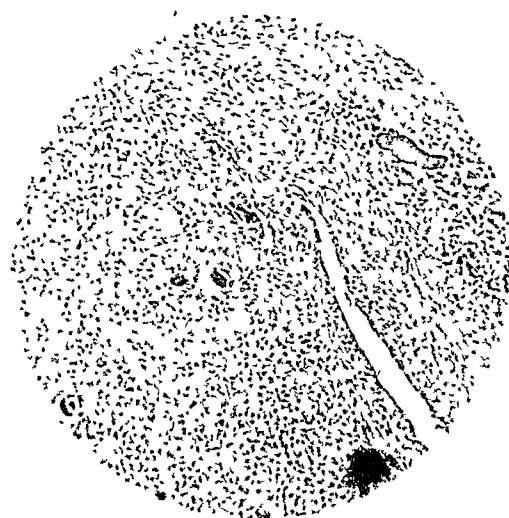


FIG. 3. High-power photomicrograph of tumor.

the tumor caused bone destruction requiring amputation of the limb.

Clinical Data. In Broders' series of 17 cases 11 were in males. The average age was forty-seven years, the oldest being sixty-five and the youngest twenty-one. Ten cases had involvement of the upper extremity and 7 of the lower. Of the former, 8 cases involved the fingers and hand of the right upper extremity and 1 of the left. Of the latter, 6 involved the right lower extremity. Ollerenshaw¹⁰ reported symmetrical tumors of the tendo Achilles on both sides in a girl of eighteen. Of 54 cases collected by Tourneaux¹¹ 8 were in the lower extremities, 5 on the toes and 3 in the malleolar regions.

Treatment. Excision is the routine procedure, and is usually easily accomplished. Amputation has rarely been necessary.

Prognosis. Of 15 cases traced by Broders 14 were living three and one-half years after operation, 1 patient having died of another condition. Ollerenshaw's case showed no recurrence three years after operation. Metastases do not occur although local recurrence is sometimes found because of incomplete removal.

June 16 he had been hit on the back of the right hand by a pair of pincers. He sustained a few scratches around the knuckle of the index finger and a swelling appeared on the palmar surface directly opposite. He had no particular pain but a feeling of weakness and uneasiness about the forefinger which persisted. The swelling did not disappear but gradually became harder. Examination on November 8 revealed a hard lump, about the size of a hazel nut and of a cartilaginous feel, apparently fixed to the proximal phalanx of the index finger. A roentgenogram taken on the same day revealed a punched-out area about 5 mm. by 10 mm. affecting the periosteum and cortex of the outer side of the proximal end of the proximal phalanx of the index finger (Fig. 1). On December 18 the patient was operated on and an encapsulated tumor the size of a hazel nut was shelled out. The specimen was of a yellowish coloration, firm and elastic in consistency. On section the cut surface was for the most part fibrous in nature, white in color with yellowish areas. Microscopically the tumor was made up of large numbers of pinkish spindle-shaped cells with pale nuclei and a pinkish staining cytoplasm. The cells were somewhat of a fibroblastic type and in places there was a considerable number of collagen fibrils laid down between the cells. Scattered throughout the whole tumor were numerous

foreign body giant cells. There were no mitotic figures present (Figs. 2 and 3). The diagnosis by Dr. W. L. Robinson confirmed by Dr. Ewing



FIG. 4. Radiograph taken July 27, 1927, showing almost complete re-ossification of bone defect.

of New York was giant-celled tumor, probably of tendon sheath. The patient was given 1 suberythema dose of roentgen therapy over the site of the tumor anteriorly and posteriorly using 103 kv., 5 ma., 5 mm. aluminum filtration, and 40 cm. distance. When seen recently, twenty months after operation, there was no sign of recurrence and roentgen examination showed complete healing of the bone defect (Fig. 4).

COMMENT

Of interest is the comparative rarity of this tumor. Up to 1913 only 54 cases had been reported. Broders in 1919 reported an additional 17 cases. Janik⁷ in 1927 was able to collect only a total of 70 cases from the literature.

Involvement of bone by a tumor of this type giving the appearance of malignancy is rare. As far as we could find, with the exception of Broders' case, this is the only reported instance of such an occurrence. The bone destruction in our case was obviously the result of pressure and not of infiltration.

Finally it may be noted that in this case as in the majority of such cases, there was a previous history of trauma. All who have studied this type of tumor agree that trauma and occasionally infection very frequently play an etiological rôle. Usually there is a clear-cut history of an accident and of its relationship to the succeeding tumor as in this case. And yet it would seem that compensation boards are slow to recognize this fact. In this particular case no compensation was awarded to the patient in spite of efforts to place the matter before the board in its proper light.

SUMMARY

A case is reported of giant-celled tumor of the tendon sheath of the finger. Such tumors are of comparatively rare occurrence. This case is of particular interest in that it caused bone erosion by pressure. Such cases often give a clear-cut history of antecedent trauma which is believed to play an etiological rôle.

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CYST OF THE PROSTATIC UTRICLE*

A CAUSATIVE FACTOR IN PRODUCING IMPOTENCY

SAMUEL LUBASH, M.D.

NEW YORK

MORGAGNI in 1742 first described cyst of the prostate and urethra to his class in the anatomy room. But it remained for English in 1873 to demonstrate first the condition now recognized as a cyst of the prostatic utricle. Following this work, Springer in 1898 in 600 autopsies on males found 4 such anomalies. It was as recently as 1915 that Klotz first demonstrated this condition in the living. Belfield then described these congenital anomalies as remains of the embryonic structure such as non-obiterated Müllerian ducts. Since then Wesson in his exhaustive study of the literature found 59 cases of cyst of the prostate and urethra reported to date. Of this number 33 were cyst of the prostate. Of those remaining, 20 were divided between cyst of the urethra and adnexa, leaving only 6 cases of true cyst of the utricle. Of these, only 4 were found in the living. Michailow in 1906 in reporting his case called attention to its association with sexual disturbance. This element was not found in the cases reported by Greenberg in 1924. In adding our case to the literature we do so not so much because of its rarity but because first, we have been successful in photographing this anomaly and second, it apparently was the cause of impotency in this particular instance.

CASE REPORT

C. U., aged twenty-five, was referred to me in November 1926 for the treatment of impo-

tency, from which he suffered untold mental anguish. His previous history was of interest inasmuch as he was quite potent up to two

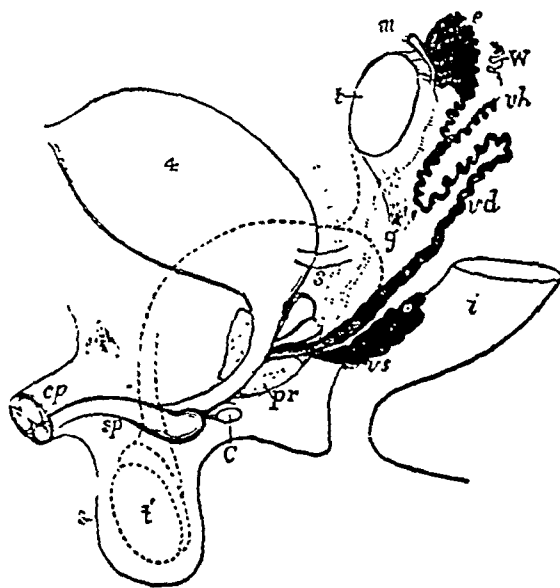


FIG. 1. Prostatic utricle.

years before coming to my attention. He had never suffered any venereal diseases. A physical examination was negative with the exception of a mass found by rectal examination in the mid-line just above the prostate. It was quite cystic to touch, and of the size and consistency of the human eyeball. Massaging of this mass had no effect on its size. A cystourethroscopic examination was performed. The bladder and bladder neck were normal, the posterior urethra was well outlined and its parts easily recognized. There was no clue as to where the mass had its outlet. Nevertheless, we passed a No. 6F. catheter into the sinus pocularis and, New York Academy of Medicine, January 18, 1928.

* Read before Section of Genito-Urinary Surgery.

much to our surprise, noted an immediate flow of a milky white fluid from the catheter. In all, about 8 c.c. was drained off. Some of this

Attempted coitus was successful for the first time in two years. To the present day his condition is most satisfactory.

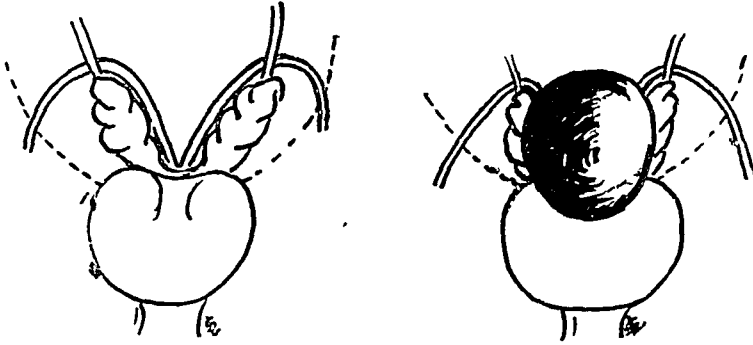


FIG. 2. Position and size of cyst posteriorly.

we lost before our collection was begun. A rectal examination now proved that the mass had disappeared. A definite diagnosis of cyst of the prostatic utricle was then made. To prove this contention a utriculogram was made which is seen in Figure 3. A cytogram and a urethrogram definitely cleared any suspicion

McCarthy in his recent contribution has well described the utricle as a sacculation or large duct occupying the central portion of the verumontanum, varying in size and shape but being approximately 6 mm. long. The actual size of the one just described

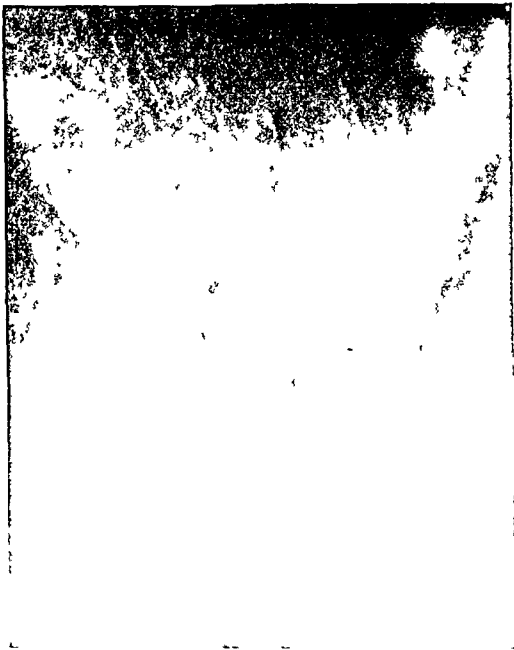


FIG. 3. Cyst of prostatic utricle.

as to the possibility of the presence of a diverticulum of either bladder or urethra. An examination one week later by re-catheterization of the utricle showed that the sac had not refilled. The patient had already experienced a slight erection. Three weeks following this he was again examined and found normal.



FIG. 4. Cystogram; negative for diverticulum.

is not known, but the measurements of the partly distended sac are 2.5 cm. by 2 cm. Comparatively this is larger than those cases reported by Michailow and Greenberg whose measurements were 1.5 cm. and 2 cm. for their respective cases.

Embryologically the utricle is the remains of the caudal ends of the old Müllerian ducts, which united to form a blind pouch with an opening into the posterior urethra. This is well demonstrated in Figure 1. Young points out that the commonest anomalies of the prostatic utricle are those associated with the various degrees of pseudo-hermaphroditism. And he quotes a case coexisting with cryptorchidism found on the autopsy table. The lesser degrees of persistence of the Müllerian duct may occur in individuals having no external abnormality of the genitalia. This was true of the case just reported.

Treatment. We know of no particular treatment in these cases. Klotz was the first to report a cure by endoscopic rupture. Many such conditions most likely go unrecognized due to spontaneous rupture, as in the case reported by Greenberg. Non-operative methods are the ones of choice. And of these catheterization should be attempted before fulguration. If the sinus pocularis is dilated sufficiently, there should be no fear of a recurrence. However, if such does happen, it is a simple procedure to recatheterize. The possibility of injecting caustics into the sac to bring

about shrinkage is suggested. We have had no experience with it, but mention it for what it may be worth.

Report of examination of cyst contents

Quantity	5 c.c.
Appearance	Milky white watery solution, which upon standing deposited a slight nebulous sediment.
Reaction	Very feebly alkaline.
Chemical analysis	Chiefly sodium chloride, mucoid substance, a trace of urea and organic salts.
Microscopic Picture	Cuboidal epithelial cells, granular cells, and partly fat-degenerated epithelium and granular matter (detritus).
Spermatozoa	None
Bacteriology	Negative

FIG. 5. Report of examination of cyst contents.

CONCLUSION

1. Cyst of the prostatic utricle probably occurs more often than one is led to believe from the literature.
2. These cysts have a definite bearing in bringing about impotency, either relative or complete.
3. Catheterization of the sac contents via urethroscopic manipulation is the method of choice.
4. Recurrence may occur.

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CASE REPORTS BY DR. PAUL W. ASCHNER*

NEW YORK

UREMIA FOLLOWING NEPHRECTOMY; CONGENITAL HYPOPLASIA OF REMAINING KIDNEY

ON February 8, 1928 a girl aged twenty was admitted to the Urological Service of the Brownsville and East New York Hospital (no. 26660) complaining of nausea, vomiting, weakness and loss of weight.



FIG. 1.

Her right kidney had been removed in November, 1927 at the Brooklyn Jewish Hospital, for an infected hydronephrotic kidney containing numerous small calculi. Three weeks after her discharge, recovery having been uneventful, nausea and vomiting necessitated readmission to that hospital. After appropriate blood and urine tests, hot packs were employed for two weeks without effect. She had lost 20 pounds in eight weeks before coming under my observation.

She appeared bright, alert, and well oriented. The conjunctivae were injected and the lower lids somewhat puffy. The tongue was dry and coated. The complexion was a very light coffee tint. Physical examination was otherwise negative. The operative scar was perfect. The left kidney could not be felt, nor was its site tender. There was no fever.

Blood pressure was systolic 130, diastolic 90. The blood showed a moderate secondary anemia. Hemoglobin was 60 per cent; erythrocytes 3,400,000; leucocytes ranged from 12,200 to 13,600; polymorphonuclear leucocytes from 74 to 85 per cent.

The blood chemistry showed urea nitrogen 150 mg., creatinine 15 mg., per 100 c.c. These figures remained constant. The blood sugar rose from .098 to .166 per cent in one week. Uric acid was 7.5 mg. on admission.

The urinary output ranged from 120 to 300 c.c. per day, despite hypodermoclysis, intravenous glucose therapy, and phlebotomy followed by transfusion. The specific gravity was 1016 to 1018, there was an intense albuminuria and acetonuria. On microscopic examination a few leucocytes and erythrocytes were found, but *no casts*. No phthalein was excreted. The eye grounds showed no changes.

Roentgen examination showed no calculi, the left kidney appeared small. On one occasion it could be palpated and felt very small. Cystoscopy revealed no obstruction, no retention in the left kidney pelvis.

Under observation certain phenomena of clinical interest developed, namely; A firm tender enlargement of the thyroid gland; a tender enlargement of both submaxillary salivary glands (probably due to hyperexcretion of urea); a diminution in the size of the liver. On the sixth day the face became edematous, the conjunctivae much injected, the tongue thickened. Twitchings of the hands and feet and one general convulsion also occurred. On the fourteenth day convulsive seizures recurred. The patient lapsed into coma, aphthous stomatitis developed and she died seventeen days after admission.

The differential diagnosis rested between an unsuspected preexisting chronic glomerulonephritis (postscarlatinal?) and a congenital hypoplasia of the remaining kidney. Surgical kidney had been definitely excluded. Glomerulonephritis could be excluded by the high specific gravity of the urine, the absence of casts, the absence of eye-ground changes. Congenital hypoplasia was therefore the diagnosis entertained.

The kidney was obtained for examination. It weighed 35 gm. The pelvis and calyces were normal in shape but somewhat smaller than normal in size. The ureter was relatively large in caliber. The capsule stripped easily and smoothly. The kidney parenchyma measured only 1 cm. in thickness at its widest point. It

* Read before the Section of Genito-Urinary Surgery, New York Academy of Medicine, October 17, 1928.

presented on the surface numerous small smooth elevations which histologically proved to be areas of hypertrophy and hyperplasia. The glomeruli were strikingly large in size and showed no evidences of inflammation or vascular changes except congestion. The tubules showed degenerative changes.

This case is reported to point out again one of the most treacherous types of congenital anomaly—a hypoplasia of one kidney which is qualitatively perfect but quantitatively incapable of sustaining life when its mate is removed for some condition requiring nephrectomy. The preoperative investigations were made by a competent observer, the operative indication was clear.

Suspicion of the condition may be obtained by a good roentgenogram showing a small kidney outline, and a pyelogram showing a pelvis which although smaller than normal is surmounted by a narrow rim of kidney tissue. In post-hydronephrotic atrophy the calyces remain large and blunted, and cystoscopic findings give evidences of much reduced renal function.

If we compare the urine put out by a hypoplastic kidney with that of its mate, the latter being normal or only slightly impaired, the urea concentration and intensity of dye excretion of the former may equal that of the latter, but quantitative estimations for a given period of time will reveal a marked difference. If however, the larger kidney of the two is greatly diseased and its function much impaired, the urea concentration and dye intensity will be so much greater on the side of the hypoplastic kidney that no suspicion of the situation will occur to the cystoscopist.

RENAL CALCULUS CONTAINING XANTHIN

The patient, a woman aged fifty, was admitted to the urological service of the Brownsville and East New York Hospital (no. 26434) for pain in the right flank, frequency and burning on urination. The symptoms were of ten weeks duration.

The urine contained traces of albumin, many clumps of leucocytes, and few erythrocytes. The phthalein output was 45 per cent in two

hours. Blood urea was 13.5 mg., creatinine 1.5 mg. per 100 c.c.

Roentgen examination was difficult as it was found impossible to free the colon completely of gas. The first film showed a very irregular shadow of somewhat triangular shape measuring 2 cm. by 1.5 cm., in the right kidney area. On cystoscopic examination the urine from the right side contained clumps of pus cells and the phthalein output was 5 per cent in fifteen minutes. The urine from the left side was normal and the phthalein output 12 per cent. The tip of the opaque catheter was in contact with the calculus. Pyelogram showed a moderately dilated pelvis. The uppermost calyx appeared large but poorly filled, the defect evidently being caused by the calculus lodged within it.

Operation was performed under gas oxygen anesthesia. The kidney was delivered and a posterior pyelotomy incision made. The tip of the triangular-shaped stone was felt projecting into the pelvis from the uppermost calyx. With the finger the narrow neck of the calyx was stretched or torn and the stone removed. In the corresponding secondary calyces three small slate-colored stones, very smooth and hard, were found and removed. The pelvis and calyces were thoroughly irrigated, the pelvis sutured and the wound closed with rubber dam for drainage. There was some postoperative leakage of urine but the wound healed well in twelve days.

I was impressed by the unusual appearance of the calculi and requested Dr. Theodor Kuttner to examine them for me. I am greatly indebted to him for the following analyses.

The small smooth gray stones contained:

	Per Cent
Xanthine.....	99.2
Calcium phosphate.....	0.6
Calcium oxalate.....	0.2

The larger irregular triangular stone which appeared crystalline with a smooth brownish gray coating contained the following:

	Per Cent
Calcium phosphate.....	69.4
Calcium oxalate.....	30.5
Xanthine.....	0.1

It is reasonable to deduce from these analyses that the primary stone consisted of calcium phosphate and oxalate and that xanthine was deposited upon the primary stone and then in the secondary calyces.

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EDITORIALS

THE LIVER AS IT FUNCTIONS AND THE OPERATION*

GORDON K. DICKINSON, M.D., F.A.C.S.

JERSEY CITY, N. J.

MEDICINE, though an art and in its theory a science, is fundamentally a philosophy and as such is affected by any discovery or alteration in viewpoint along correlated paths of knowledge, but the mind may be diverted from the essence of things for a considerable period, and, the art becoming more prominent, Nature's workings are relegated in thought.

Long and Lister having added to our armamentarium, a much needed advance in therapy soon prevailed to the great benefit of the race. But the present generation has seen little in literature save how to act. It is now time for a return to the fundamentals. We should learn

how to think. The division of medicine into two sections, surgery and internal medicine, is irrational and can but subvert the truth.

A century ago Bichat taught the need of studying the life history of the cells. He taught cell action typifies structure action and there from we must obtain a true knowledge of the activities of each organ and its correlations. Progress in medicine still depends upon an accurate knowledge of cell life.

Unmindful of the rest of the body, the average practitioner conceives of disease as a local process, that the viscera act "each for itself." The keen, broad-minded practitioner recognizes no strictly local

* Read at the forty-first Annual Meeting of the American Association of Obstetricians, Gynecologists and Abdominal Surgeons, Toronto, September 10, 1928.

pathology, knowing that if one part ails all join in a sympathetic reaction, even to the remote parts of the body economy, "each for all and all for each."

As is the liver cell, so is the liver, and as is the liver, so is life, physically and mentally. No cell or colony of cells has so far-reaching an effect on comfort and distress, either immediate or remote. The ameba, a familiar type of single cell life, meets all its own biologic demands, yet is simple in structure. Most of the body cells have a particular function and show a special morphology, but the liver cell remains primitive, an ameba *hepar* in its simplicity, possessing the possibilities of many functions, seventeen in number. No highly organized cell can accomplish this.

Its primitive habits remain latent in it. The embryonic urge leads each tissue and organ to grow until a maximum is reached, then cease, a law as immutable as gravity or heliotrism. Remove a section of the liver, a third, for example, and the ameba *hepar* begins to act like its progenitor, to divide and reproduce until an amount necessary for the system is attained. The liver is very slow to develop. Primarily of tubular structure it is not until the eighth year that the lobules are fully formed. It is kept safe from trauma and chill high up under the dome of the diaphragm, with a temperature about two degrees F. above the accepted norm.

The liver is unique in having two blood supplies: the usual arterial red blood with high pressure through a comparatively small lumen with arterioles capable of independent contraction, and a venous blue blood with almost negative pressure through a vessel ten times larger. The latter goes directly to the active cells, while the former, being mainly for the parenchyma, ends by emptying into the portal radicals before they bathe the liver cell. Here the circulation is very slow and activated by pressure from below and the suction effect of the diaphragm. Sixty per cent of the blood flowing through the liver is from the portal vein.

All the activities of each cell are conducted and maintained through carbonized blood, a negation to all the other tissues of the economy. If electricity be the potent force of cell action, what may it be here, and what may disturb where so much is reversed?

The nerves of the liver are three fold: (1) the sympathetic, the nerve of concord and harmony, subject to fear; (2) the pneumogastric, the more alert in apprehension, passing warnings to the basal centers of the sympathetic to have care and to act; (3) the phrenic, of unknown value, except perhaps when irritated to splint the diaphragm. Nonmedulated fibres have recently been found to pass into each lobule and alongside each cell.

The mesh of cells surrounding the central vein, the hepatic radical, are as a sieve for the blood which flows very slowly. The outer cells seem to obtain more sustenance than the inner, that is, more oxygen. As a result, the inner cells are less resistant to chemical and nerve violence. They may show changes after any prolonged anesthesia. The hypothetical histamin from wounds, toxins from the gut and pregnant womb, sensitizing proteins and even alcoholics, all carry danger possibilities. Fortunately, stored glycogen is an efficient protector, and a well-fed and well-slept man does not succumb readily. An empty cell cannot detoxify and is subject to necrobiosis.

Even with an hepatic cell distended with glycogen, it can retain amino-nitrogen up to 150 per cent. This is released by a different method than the glycogen. The blood needs only a certain amount of the latter but seems to require much of the former, the aminonitrogen, which may account for the fact that vegetable eaters win the Marathon and the meat eaters the sprint.

Another function of importance to the surgeon is the role the liver plays in acidosis, a break in the formation of urea resulting in such a condition. It is the faculty of every tissue of the body to rid the blood

of impurities, but the liver has the fecal blood brought to it from the intestines, and according to its glycogen content is it successful. Purges increase the foulness of this stream and diminish insomuch the content of glycogen.

Clinical and historical medicine strongly indicate that the more recently developed cerebral tissues are fed through or by the hormone of this primitive organ. Not to assert that all the other organs of the body are less important in function, but the liver, being the oldest biologically, is of more special importance. The Babylonian cryptic has not as yet been solved, and the liver still remains an organ of mystery. Besides the seventeen functions ascribed to it there may be others. Being a primitive tissue each cell in it is activating itself in each of these seventeen ways at the same time. Several of these duties are of supreme importance to the physician and surgeon, and the man who is uneducated and who does not appreciate them is a danger.

Each and every liver cell is a constituted depot for a universal food staff, and this is the only place with such a function. After each meal of carbohydrates the starch is converted into glycogen and stored, swelling each cell enormously, the elastic and semigelatinous fibers of the capsule permitting the liver to enlarge thirty per cent or more. Insulin and the parathyroid aid the enzyme contained in each cell to produce this result. Here it remains until a demand is made for more sugar in the blood, then the thyroid and adrenals cooperate in its withdrawal through the sympathetic.

With this the vagus unites on conservation, having fibers which carry impulses centrally, which when reflected to the sympathetic through the combined efforts of the thyroid and adrenal withdraw enough glycogen to satisfy demands.

Sleep and rest of mind and body conserve glycogen, while insomnia, anxiety, and mental and physical trauma, particularly intra-abdominal, liberate it. To re-

duce it materially leaves the organs, especially the brain, in a dilemma. Impending acidosis, insomnia, restlessness and nausea result.

A conserved glycogen content means body comfort and a winning fight. For comfort and recovery a patient should rest recumbent for at least two days before any operation and should have sleep by night and naps by day. There should be no unpleasant distraction; the mind should be at ease and nursed into an optimistic frame, with forgetfulness of home and business lacerations. The patient should be fed carbohydrates plus hydromel and oxymel, that is, ten per cent glucose in water, plain or flavored, an evidence of the good judgment of the ancients.

Preliminary preparation should be simplified. Anesthesia must be expertly administered and incisions so placed that exposure of the nonpathologic area be minimized, for overexposure allows the intestinal hormone, carbonic acid gas, to evaporate, which no gauze, wet or dry, can inhibit, leading to local paresis, or so-called pseudoileus and gas pains. There should be no retracting, no packing, no swabbing, for they are all rude insults to the sympathetic and allow manifold reflex effects. Arterioles will contract, capillaries dilate, and shock, either general or local, is produced. To the stomach, acute dilatation; to gut, tympany; to liver, suppressed functioning, and as this organ feeds the brain with some unknown hormone, death may ensue. Each punch of a swab, each retraction with its slipping and readjustment, each pack introduced, results in retaliation to the insult. Glycogen is lost, and if over much, misery follows. The rude operator is brutal.

Specialization led to civilization, in turn reacting to further specialization, but civilization is artificial and may retrograde. There have been many, and all so far have passed into history. Likewise, tissue specialization may fail. Nature has made more than one alimentary

canal, more than one type of heart and has added brain on brain, but the liver is ever the same.

During these two score years out of its chrysalis state surgery has been dominated by anatomy. The mind of the surgeon lies in the scalpel, guided by a more or less perfect knowledge of the body. Is it not time now that a new and more competent guide be chosen, one that will make for comfort of patient and outlaw much unnecessary operating?

To equip a patient for an operative ordeal all undue preparation must be considered a psychic disturbance and should be avoided, for it is a drain on the chromaffins and glycogen. The operation should be performed gently, without swabbing, retracting, or packing except when needed for the protection of viscera and not for the ease of the surgeon. It should always be remembered that a touch, no matter how light, means a call on the glycogen reserve. It is difficult to make the young operator appreciate this great law, but if it be taught in college and clinic, surgery will some day lose much of its horror.

The hand and the eye cooperating through the olive have bent the mind

toward the mechanical. In ancient times the Egyptians and the Greeks developed logic, philosophy and medicine by reasoning. Now the practice of surgery is largely mechanical, with diagnostics tending toward the same. The laboratories have been of immense scientific and practical gain to medicine, but reports and shadows soon become idealized, and then false idols spring up. It seems as if we meditate less, through the training of the mind in deep thought is divine.

The liver is a most interesting study with its functions so varied and its morphology so unique. To know it is insistent, and to act with and for it is imperative. This etching has in it the seeming proofs cytologic and physiologic, that the liver and its associated organs, the thyroid, adrenal and pancreatic islands, are of fundamental importance to physician and surgeon, laboratory research and experience as well having proved it.

Again, the system needs glucose for action. Let it be given for that. It needs glucose to maintain the integrity of the liver. So give it for that. Carbohydrate diet and the drinking of 10 per cent glucose, (plain, hydromel; with fruit juices, oxymel) will make for comfort and recovery.



BRITISH LETTER

IN my last letter in which I referred to the routine methods of British surgeons generally for the treatment of ulcers at and about the pylorus no mention was made of the use of Horsley's operation by the Edinburgh surgeons, particularly Professor John Fraser, who favours this form of pyloroplasty for certain ulcers about the pylorus, one great advantage being that the ulcer may be excised and the pyloric orifice reconstructed in such a way that there can be no possibility of stenosis. Further the risk of recurrent ulceration appears comparatively less than

after a gastroenterostomy. The proper indication for the operation appears to be a small limited ulcer in the first part of the duodenum, certain types of small prepyloric ulcers, or to give rest to the stomach after the excision of a gastric ulcer in some other part of the viscus.

Fraser stated before the Manchester Surgical Society that he was well satisfied with the results of the operation after an experience with over 70 cases and that it was a valuable conservative operation if used with foresight and proper technical skill. There has been a stream of

literature on the subject of gastric ulcer lately, individual surgeons indicating individual preferences. John Morley (Manchester) impressed with Schoemaker's method has modified the Schoemaker clamp and produced an instrument which can be applied from the lesser curvature. He considers that it is time to assess the results from the many and various types of gastrectomy that are being performed, what influence they have on gastric function and on the general well being of the patient. He insists that the Polya gastrectomy does as a rule abolish gastric digestion completely. He says the food, with such little gastric juice as continues to be secreted, drops straight through into the jejunum without even a transient hold up in the stomach, and what is more serious still is that a certain proportion of patients after a Polya showed a marked tendency to anaemia which resisted all forms of medical treatment. He declares his preference for the modified Schoemaker operation. His analysis of 68 cases in his own practice leads him to consider it gives better clinical results and the patients show practically no signs of postoperative anaemia. Further it is radical in that it removes the ulcer-bearing area and the pyloric sphincter and permanently lowers the acidity of the gastric secretion, whereas it is conservative in that it leaves a stomach which functions in a manner akin to the normal organ.

Two papers dealing with fractures of the lower end of the radius have appeared recently in the *British Medical Journal*, the first by Messrs. Edwards and Clayton of King's College Hospital,¹ the second by Messrs. Grasby and Trick of Guy's Hospital.² Both record a certain number of unsatisfactory results following on the customary methods of treatment adopted at these Hospitals respectively. Some 420 cases are reviewed in the first paper and 50 cases in the second, but in this last series the observations are upon cases

of Colles' fractures sustained two years previous to the investigation and the following conclusions are tabulated:

1. Colles' fracture without displacement yields uniformly good results, as observed two years after the injury.

2. Where displacement was a marked feature of the fracture the results two years after the injury are invariably unsatisfactory.

3. The disability was in each case due to osteoarthritis in the inferior radioulnar joint, resulting from incomplete reduction.

These are the deductions of two junior surgeons, and have called forth a very interesting and diverse correspondence concerning one of the commonest forms of fracture. Yet contrary to expectation although capable of treatment in so many ways and by so many forms of splinting there has resulted a confusion of ideas, and in spite of the multiplicity of choice in the methods of reduction and apparatus of fixation it is known to be followed far too frequently with bad and indifferent functional results. Blundell Bankart makes some very forcible remarks on the subject. He writes from the conclusions given above that "we must infer that at a great London Hospital a Colles' fracture is never properly reduced, for apparently, when the displacement is not considerable reduction is not considered necessary and when displacement is considerable the result is invariably unsatisfactory owing to incomplete reduction." Further his own experiences of cases of Colles' fracture lead him to believe that hardly ever is the fracture properly reduced although many of the cases he has seen had previously been manipulated in an attempted reduction. The reason for the bad results he attributes to the fact that the Sir Robert Jones' method of reduction is invariably attempted, but inadequately and incompletely performed.

The possibility of mistaking the fracture for a sprain or dislocation is today hardly conceivable, but before the advent of roentgen rays and certainly in Colles'

¹P. 61, Jan. 12, 1929.

²Mar. 2, 1929.

time, it was by no means an unusual mistake as may be gathered from his introductory remarks to his famous clinical description of the fracture of the carpal extremity of the radius which we now always associate with his name:

The injury to which I wish to direct the attention of surgeons, has not, as far as I know been described by any author; indeed, the form of the carpal extremity of the radius would rather incline us to question its being liable to fracture. The absence of crepitus and of the other common symptoms of fracture, together with the swelling which instantly arises in this, as in other injuries of the wrist, render the difficulty of ascertaining the real nature of the case very considerable.

This fracture takes place at about an inch and a half above the carpal extremity of the radius, and exhibits the following appearances.

The posterior surface of the limb presents a considerable deformity; for a depression is seen in the forearm, about an inch and a half above the end of this bone, while a considerable swelling occupies the wrist and the metacarpus. Indeed the carpus and base of metacarpus appear to be thrown backward so much as on first view to excite a suspicion that the carpus has been dislocated forward. On viewing the anterior surface of the limb, we observe a considerable fulness, as if caused by the flexor tendons being thrown forwards. This fulness extends upwards to about one-third of the length of the forearm, and terminates below at the upper edge of the annular ligament of the wrist. The extremity of the ulna is seen projecting towards the palm and inner edge of the limb; the degree, however, in which this projection takes place, is different in different instances.

If the surgeon proceed to investigate the nature of this injury, he will find that the end of the ulna admits of being readily moved backwards and forwards.

On the posterior surface he will discover by the touch that the swelling on the wrist, and metacarpus, is not caused entirely by an effusion among the softer parts; he will perceive that the ends of the metacarpal, and second row of carpal bones, form no small part of it. This, strengthening the suspicion which the first view of the case had excited, leads him to examine, in a more particular manner, the

anterior part of the joint; but the want of the solid resistance, which a dislocation of the carpus forward must occasion [*sic*], forces him to abandon this notion, and leaves him in a state of perplexing uncertainty as to the real nature of the injury. He will, therefore, endeavour to gain some information by examining the bones of the forearm. The facility with which (as was before noticed), the ulna can be moved backward and forward, does not furnish him with any useful hint. When he moves his fingers along the anterior surface of the radius, he finds it more full and prominent than is natural; a similar examination of the posterior surface of this bone, induces him to think that a depression is felt an inch and a half above its carpal extremity. He now expects to find satisfactory proofs of a fracture of the radius at this spot. For this purpose, he attempts to move the broken pieces of bone in opposite directions; but, although the patient is by this examination subjected to considerable pain, yet neither crepitus nor a yielding of the bone at the seat of the fracture, nor any other positive evidence of the existence of such an injury, is thereby obtained. The patient complains of severe pain as often as an attempt is made to give the limb the motions of pronation and supination.

This is the description which Abraham Colles, M.D., one of the professors of anatomy and surgery in Ireland wrote for the *Edinburgh Medical and Surgical Journal* in 1814 and compared to the meagre descriptions that appear in many modern text books it may rightly be considered a masterpiece of lucidity.

Much has been added to our knowledge of this type of fracture since the introduction of roentgen-ray examinations and we are aware of many varieties of fractures of the lower end of the radius to which other names have been subscribed, but of these the unreduced impacted Colles' is probably the most disabling and hence the importance of early and adequate treatment.

The nature and methods of reducing the deformity are legion. Colles' original method was: "Let the surgeon apply the finger of one hand to the seat of the suspected fracture and, locking the other hand

in that of the patient, make a moderate extension until he observes the limb restored to its natural form." This is of course a method of traction which in many cases requires more force than is here implied, particularly as many cases are still seen late and require forcible interference to obtain proper correction. For these cases as Bankart says the Jones' method so much used in this country is not sufficient in the hands of the occasional manipulator, and he advises forcible reduction over a wedge, and thus describes the procedure:

The patient must be fully anaesthetised. Some expert anaesthetists can give gas and oxygen for long periods, and even can obtain muscular relaxation (with luck) but this is quite exceptional. As ordinarily given "a whiff of gas" is entirely inadequate for the proper reduction of a Colles's fracture. In these circumstances the reduction is always hurried, the patient is usually stiff or moving, and, above all, there is no time for a critical review of the reduction before he comes round, nor for a repetition of the manipulation if it should be found to be incomplete. The reduction should be done deliberately with the patient fully anaesthetised, I think that regional (brachial plexus) anaesthesia might be employed here much more often than it is.

For the manipulation the fracture is laid flexor surface downwards across the wedge, so that the lower end of the upper fragment rests directly on the wedge. For the right side it is convenient to have the wedge lying on the table by the patient's side; for the left side (unless the surgeon is left handed) it is more convenient to have it on an outlying small table, the upper fragment is firmly held by the surgeon's left hand on the wedge. The lower fragment is gripped by the surgeon's right hand with the thumb on top and the ball of the thumb directly over the prominence which the displaced lower fragment makes on the dorsum. The lower fragment is now forced downwards and forwards and towards the ulnar side. The amount of force required varies in different cases, but in all cases is considerable, and I have many times put the whole weight of my body on to this fracture in order to reduce it properly. At the end of the manoeuvre the backward tilt of the lower

fragment is corrected by a sharp forward flexion movement.

The severity of this measure has been criticised by Sir Robert Jones, Max Page and C. H. Fagge, who consider there is a great risk of seriously injuring the nerves and tendons lying between the bone end and the wedge, but in Bankart's description there seems to be no suggestion of damaging these structures. Another point raised is the question of the best way to maintain the position after reduction for although there may be little tendency in some cases for the deformity to recur in others a misapplied or unsuitable splint may easily displace the fractured ends. For instance to use a dorsiflexed splint as suggested in a recent textbook is a procedure which will tend to cause a return of the displacement. The contention as to whether the arm should be put up in pronation or supination is not of such vital importance if one takes every care to see that no splint is applied which will tend to press the lower fragment backwards. Without being dogmatic in any way the most comfortable splints are the properly twisted malleable iron splints and well padded devised by H. O. Thomas and applied as follows: The posterior splint extends from the external condyle of the humerus to about the middle of the metacarpals, and runs spirally from the ulnar side at the elbow to the radial side below. The anterior splint on the palmar aspect of the forearm has the twist in the opposite direction and must stop short of the thenar eminence. Fixation is maintained by strapping and a bandage, leaving the fingers free. Otherwise a plaster gutter splint moulded to the flexor surface of the forearm and hand leaving the fingers free is equally satisfactory and has the virtue of cheapness and accessibility. The plaster splint to be effective will be applied with the hand and wrist swung round into position of palmar flexion pronation and ulnar deviation (Cotton-Loder position). Finally the anatomical reduction must be verified by a roentgen-ray examination.

The after treatment is of great importance; finger movements are encouraged from the beginning and movements of wrist joint initiated in two to three weeks' time. Fixation is maintained for four weeks, afterwards active movements of the wrist and radioulnar joints are attempted and resistive exercises for the fingers persevered with. Massage and arm baths will aid considerably in hastening the return of function and power.

CAMPAIGN AGAINST CANCER

Since the International Conference on Cancer held in London last July a great impetus has been given to the campaign against cancer in this country. The Government has been stimulated at last to act and as a result a Radium Subcommittee was appointed. The findings of this body are now published and a series of recommendations have been made to the Government which are embodied in the report just issued by H. M. Stationery Office (April, 1929). After a brief account of the development and elaboration of the Radium Technique the Committee reaffirm the view now generally held that radium will in increasing measures replace excisional surgery in the treatment of cancer. Stress is laid on the inadequate amount of radium available in England, the shortage

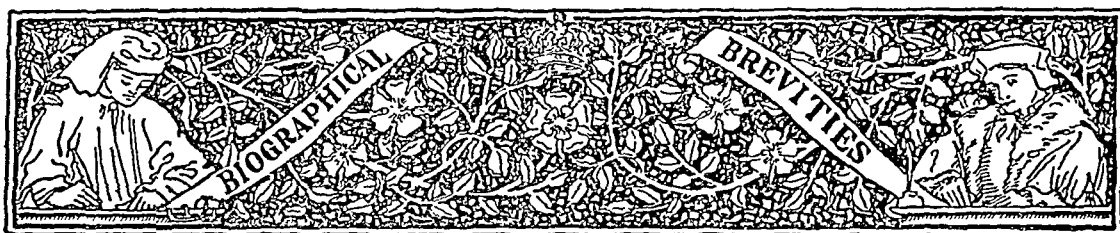
of highly trained radium surgeons, and the corresponding lack of hospital beds. At present there appear to be only 10.667 grams of radium in the whole of Great Britain, which, of course, in the opinion of the Committee is far from being sufficient for the requirements of the population. They, therefore, recommend the immediate purchase of another 20 grams of radium, and suggest that the funds required be obtained in part by public subscription, and that the Government be asked to contribute pound for pound up to some maximum sum to be determined. This maximum sum is now declared at £250,000 and the Government has agreed to give £100,000.

This fund will be under the control of a Radium Commission of experts who will organise a policy for the distribution and use of the radium thus acquired. The Radium Commission is not intended to attempt to secure complete control and centralisation of all radium in the country. Individual institutions will continue to hold their own stocks. But every endeavour will be made to coordinate the distribution for the use of the various areas of the country so that the radium may be used continuously, efficiently and to the best advantage.

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Subscribers to THE AMERICAN JOURNAL OF SURGERY visiting New York City are invited to make the office of the publishers (Paul B. Hoeber, Inc., 76 Fifth Avenue, New York) their headquarters. Mail, packages or bundles may be addressed in our care. Hotel reservations will gladly be made for those advising us in advance; kindly notify us in detail as to requirements and prices. List of operations in New York hospitals on file in our office daily.



CIRCLE OF WILLIS

THOMAS WILLIS was the son of a Wiltshire farmer. He was born in 1621 and lived for fifty-four years. Into that time he crammed the achievements of several average lifetimes.

Willis graduated from Christ Church College in 1639 and was Sedleian Professor of Natural Philosophy at Oxford in 1660.

In 1666 he moved to London and soon became the physician to those comprising the fashionable world of the day. His practice was enormous and his income large.

In addition to these activities he was a voluminous writer. Among his more important works were "Cerebri Anatome" (1664), "London Practice of Physick" (1685), "Pathologiae Cerebri, et Nervosi Generis Specimen" (1667), and "Pharmaceutice Rationalis" (1674) which gives one an accurate epitome of the materia medica of the time.

Willis is said to have made the best qualitative analysis of the urine possible in those days, and was, also, the first to notice the sweetish taste characteristic of diabetic urine. He described the Erb-Goldflam symptom-complex. Willis gave the first account of epidemic typhoid fever. He was the first to describe and name puerperal fever.

A great clinician, in his "Pathologiae Cerebri, et Nervosi Generis Specimen" he gave to posterity many accurate, detailed clinical pictures. In the light of later knowledge his reasoning about the physiology of the nervous system was, very often, away off key. This has caused many writers, ever looking for flaws, to overlook his genius as a truly great clinician.

Probably his greatest and most enduring work was "Cerebri Anatome," illustrated by Sir Christopher Wren. In this work Willis gave the most complete and accurate account of the nervous system which had appeared to that date (1664). It contained the classification of the cerebral nerves and gave the first description of the 11th cranial nerve or "nerve of Willis." Also, he described the hexagonal network of arteries at the base of the brain, now known as the "circle of Willis," with which every medical student at some time conjures, and which anatomical label is responsible, among the majority of the profession, for any acquaintance with the name.

Willis, still a man in the ripe fullness of his years and in the midst of his many-sided vocations and avocations, died in 1675.

T. S. W.





THOMAS WILLIS
[1621-1675]



[From Fernelius' *Universa Medicina*, Geneva, 1679.]

BOOKSHELF BROWSING

AN OSLER MANUSCRIPT



FIG. 1. Ambroise Paré at the age of forty-five.

[We are publishing a facsimile reproduction of the Sir William Osler's holograph manuscript on the "*Anatomie Universelle*" of Ambroise Paré. The essay is reprinted by permission from the *Annals of Medical History*, Vol. 1, No. 4. Ed.]

THE early works of the Father of French Surgery were in the vernacular, and so popular that, like school books, they were "thumbed" away, and few copies remain. Among the rarest is the "*Anatomie Universelle*" of 1561, of which Malgaigne knew of only two copies—one imperfect in the St. Geneviève Library and the other at Bar-le-duc in private hands. The St. Geneviève Library is the fortunate possessor of six of the nine works which preceded the great surgery of 1575; but the *Anatomie* is not at Washington, nor in the British Museum or Bodley, nor, so far as I can ascertain, in any of the special collections, except the Hunterian at Glasgow. Dr. Hahn writes (1918) that it is not in the Bibliothèque Nationale or in the library of the École de Médecine.

Not long ago in a Paris catalogue, a copy was advertised, and after a hurried look at Malgaigne and the Index Catalogue of the St. Geneviève Library, I sent a telegram and was delighted to get the book within forty-eight hours. The provenance is uncertain. It had come in with a number of unbound volumes and was sent to Chambolle-Duru, in whose famous morocco and unmatched gilding it is now adorned—a small octavo of 277 pages. The work must have been a boon to the surgical students of St. Côme, very few of whom, like Paré himself, had had a classical training. Both editions of the great "*Fabrica*" had been

The Anatomie Universelle of Ambroise Paré. The early works of the Father of French Surgery were in the vernacular, and so popular that, like school books, they were 'thumbed' away, and few copies remain. Among the rarest is the Anatomie Universelle of 1561, of which Malgaigne knew only two copies, one imperfect in the St. Genievre library and the other at Bar-le-Duc in private hands. The S. G. L. is the fortunate possessor of six of the nine works which preceded the great surgery of 1575; but the Anatomie is not at Washington, nor in the British Museum, or Bodley nor, so far as I can ascertain, in any of the special collections, except the Huxleyan at Glasgow. Dr. Huxley writes (1918) that it is not in the Bibliothèque Nationale or in the library of the École de Médecine.

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The gem of the work is a copper engraving, the

published, and the text and plates, particularly the latter, are largely Vesalian. figure 5 changed to 8. It is by far the most pleasing picture, and I have not found in the Hope and other collections

2 earliest known portrait of Paré, ^{aged 45;} in a wood-cut which, an oval medallion, appeared a few months later in La methode curative des playes &c.. Both bear the legend Labor improbus omnia vincit, in the latter work encircling the picture, not at the base. It was reproduced in the Dix livres de la Chirurgie, 1564, with the figure 5 changed to 8. It is by far the most pleasing picture, and I have not found in the Hope and other collections available a reproduction. The impression is unusually clear, much more so than the copies from La methode curative and the Dix livres just referred to. The great surgeon is here seen in his prime, and one may read in the face "the gentle masterly and true man" (Allbutt). The fitness of things demands that ~~my~~ this copy should return ultimately to France, to the great collection of the École de Médecine.

William Osler

FIG. 2B.

FIG. 2A and B. Osler Manuscript. (In possession of Paul B. Hoeber.)

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WILLIAM OSLER.

ANAT IE VNIVERSELLE DV

Corps humain, composée par A. Paré Chirurgien ordinaire du Roy, & Juré à Paris : recueüe & augmentee par ledit auteur avec I. Rostaing du Bignonc Pro-uencal aussi Chirurgien Juré à Paris.



A PARIS.

De l'Imprimerie de Jehan le Royer, Imprimeur du Roy és Mathématiques, demeurant en la rue S. Iaques, à Penfeigne du Vray Potier, près les Mathurins.

AVEC PRIVILEGE DV ROY.

1561.

FIG. 3. Title page of "Anatomie Universelle."

[Sir William's copy of "Anatomie Universelle" was on his death duly presented to the Bibliothèque Nationale, and a few days later a copy was picked up for a few francs on one of the famous bookstands on the Quai. There are now known to be several other copies in public libraries and in private hands. There is an illustration of the title page of this volume in "A Catalogue of Manuscripts and Medical Books Printed Before 1640, in the Library of LeRoy Crummer, Omaha, Nebraska" with the caption "The Rarest Medical Book in the World."]

BOOK REVIEWS

One of the best single volumes on medical history extant¹ by one of America's greatest medical historians has now appeared in its fourth edition and, according to the author's preface, "for the last time." This statement should be subject to modification, for the improvements in this edition are so great that it would be splendid if the distinguished author could be induced to furnish revisions every few years for many decades (incidentally, it appears from the copyright that the third edition appeared in September, 1921, whereas, in the preface to the fourth edition mention is made of "the last edition 1922"). As Garrison says regarding the changes in viewpoint which have been effected in the fundamental disciplines of medicine: "To render a clear account of these is no easy matter; to deal adequately and exhaustively with nuances of such complexity would require whole-time labor over a number of years, in a work of several volumes." Though he adds that: "for such a task the author has neither the time nor the inclination" he should be induced to "change his viewpoint" for no one is better equipped to write or edit such a work than Colonel Garrison. How so much information was condensed into the narrow limits of one thousand pages is bound to be a source of wonderment to every reader. The amount of learning both factual and critical at the disposal of the author is something before which the student of medical history must stand aghast. As a book of reference this volume is indispensable to every physician. Little is left unanswered in its pages and the unusually large number of references render the procuring of further details a simple matter. As a New Yorker the reviewer is glad to see the elder Janeway included in this edition. This had been one of the names previously found lacking. Misprints are inevitable in a work of this kind. That there are so few is evidence of the infinite care taken by the author and the publishers. The "Chronology of Medicine and Public Hygiene" of seventy pages is alone worth the purchase price. Of course, everyone will find some of his favorite names and dates missing but after all who is better equipped than Garrison to prepare a "standard" chronology. The appendices on

¹ AN INTRODUCTION TO THE HISTORY OF MEDICINE. By Fielding H. Garrison, A.B., M.D., Lieut. Col. M. C., U. S. A. Ed. 4, Phila., W. B. Saunders Co., 1929.

"Hints on the Study of Medical History" and "Bibliographic Notes for Collateral Reading" with a Bibliography of 36 pages are invaluable. The numerous references to modern American contributions to medical history largely quoted from the *Annals of Medical History* are a tribute to the increasing interest in this country in the cultural side of medicine. Seven pages of "Questions and Exercises" precede the index. Here the author succumbs to the modern "Ask-me-another" fad and a glance through these particular pages leaves one with rather mixed feelings. The subjects run the gamut of medical history and range from profound problems like "What was the Hindu method of teaching surgical procedure" to such trivialities as "Translate into idiomatic English 'Hyrtl's address over a box of cigars.'" Who, but our author, can answer offhand a series of such posers as "Why did Goethe dislike Haller?" "In what subjects did the Arabian physicians excel?" "Why did *Life* (New York) satirize medicine during the decade 1908-1918?" "Why did Naunyn decline the chair of internal medicine at Vienna?" "What pathological lesions have been found in Egyptian mummies?" etc. There are one hundred and twenty-five such questions. Only a genius could answer them all. Only a genius could think them up, but then, only a genius could write this book. A splendid iconography is added to the text. The publishers modestly refrain from numbering the illustrations, and the reviewer hasn't the patience to count them; suffice it, therefore, to say that there are hundreds of pictures of medical men and medical objects. The first is a page of symbolic Grecian figures, the last a picture of Carl Sudhoff (the Garri-son of Germany) and between the two there are illustrations of practically everything illustratable.

No self-respecting library *can* be without this book; no physician with any pretense at culture *should* be without it.

Here is a splendid little manual of Minor Surgery,¹ which covers the ground completely and without padding. It is illustrated where necessary and clearly written. It is divided into 12 chapters ranging from "Preparation of the

¹ KLEINE CHIRURGIE. By Prof. Dr. Hans Kurtzahn. Berl., Urban & Schwarzenberg, 1929.

Patient" to "After Treatment." Just the book for the general practitioner to have at hand for ready reference.

The third edition of a very expensive but very practical book¹ covers those minor conditions of the feet which are usually neglected in our textbooks of surgery and on which most physicians will be glad to have the information found in this volume.

The changes for the better in this new edition² are perhaps best exemplified in the change of title, the first edition, called "Kystoskopische Technik," was published in 1923 and has been out of print for some time. The new and enlarged edition has been called "Lehrbuch der Diagnostischen und Operativen Cystoskopie," because, as he states in his preface, the author realizes that the good cystoscopist needs in addition to being a good technician to have a knowledge of functional tests, pyelography, and above all should be a good clinician. This book therefore is for the urologist who uses the cystoscope as he does other diagnostic aids, when and where indicated. We know of no other book which treats the subject from this common-sense angle in as complete, up-to-date and withal practical a manner. This edition should be in the library of every urologist.

The author³ states in his preface that in his opinion "The reader is entitled to have developed for him a coherent story, one that impresses all the concatenated events leading up to the clinical situations with which it is the purpose of this book to deal." This intention the author has lived up to in its entirety. A more complete discussion of the diseases and deformities of the spine and thorax the reviewer has not yet discovered and this book is a decided addition to the orthopedic literature of this country. To the orthopedist it is indispensable. To every surgeon it will be of inestimable value. In make up the book leaves little to be desired.

¹ PRACTICAL CHIROPODY. By E. G. V. Runting, F.I.S.CH.

² LEHRBUCH DER DIAGNOSTISCHEN UND OPERATIVEN CYSTOSKOPIE. By Dr. Eugene Joseph. Ed. 2, Berl., Julius Springer, 1929.

³ DISEASES AND DEFORMITIES OF THE SPINE AND THORAX. By Arthur Steindler, M.D., F.A.C.S. St. Louis, C. V. Mosby Co., 1929.

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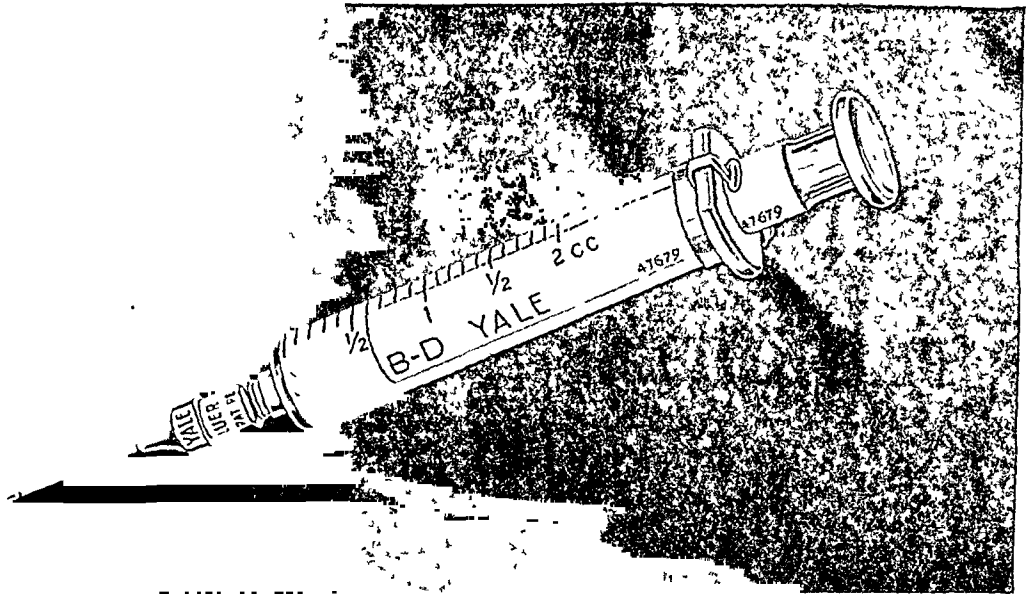
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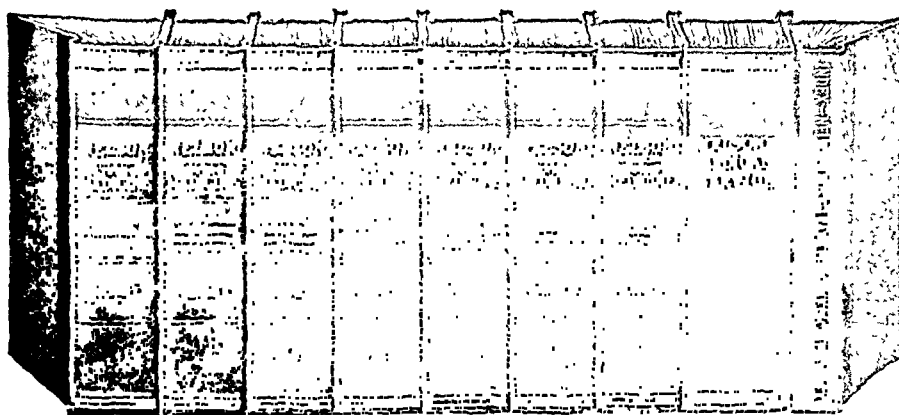
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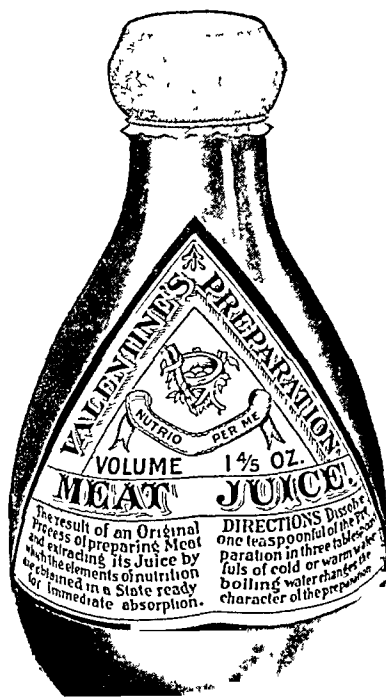
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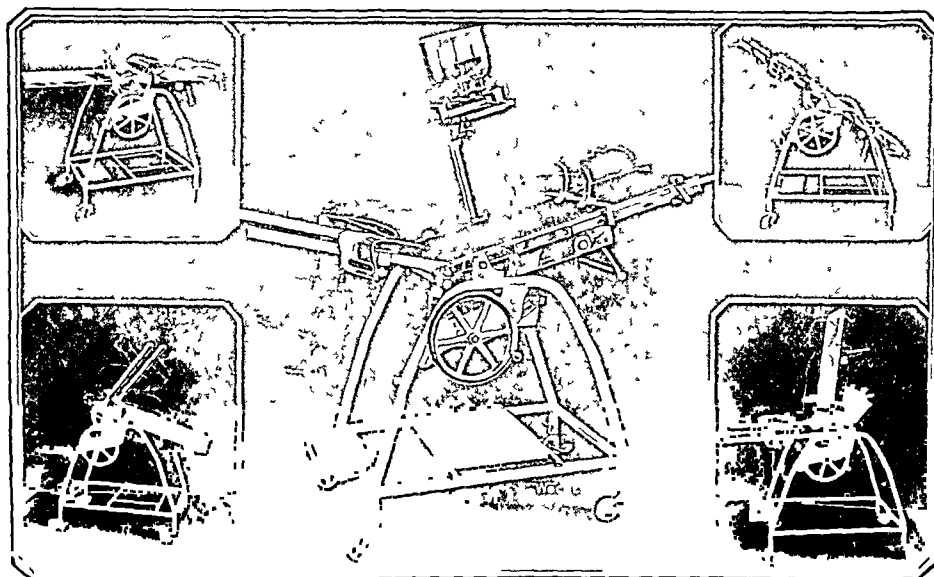
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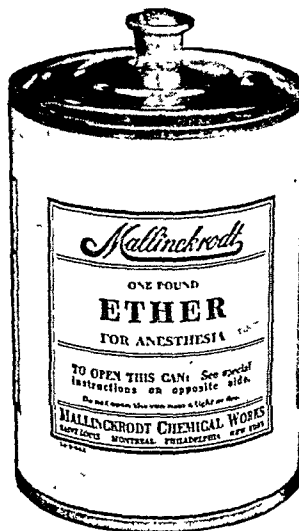
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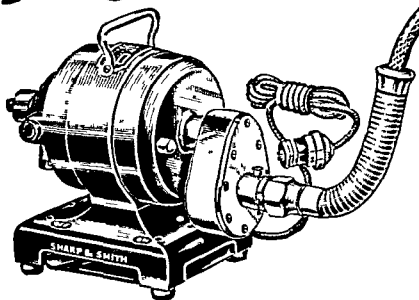
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THE book is a presentation of personal experiences; facts, and not theories, are presented; and only those facts in which it has been possible to check up the end-results. Special attention has been paid to the immediate treatment and after-care of traumatic conditions, because the author has felt the lack of this in other treatises on the subject.

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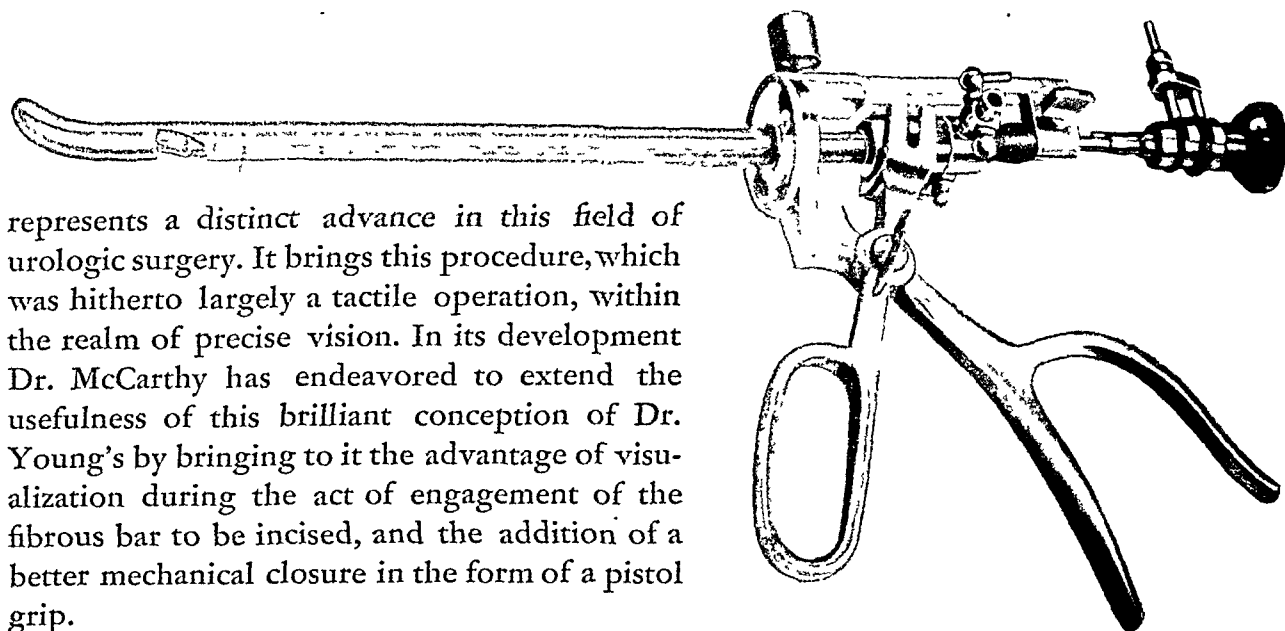
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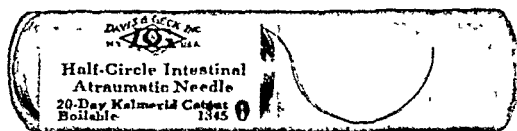
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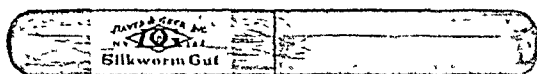
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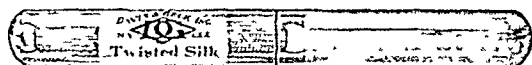


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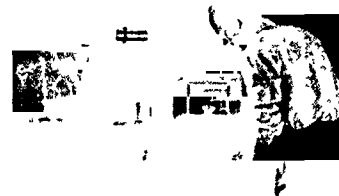


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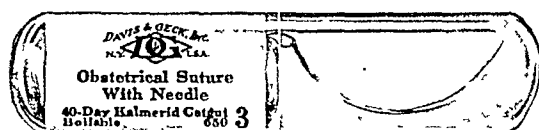
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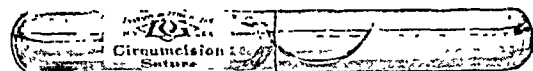
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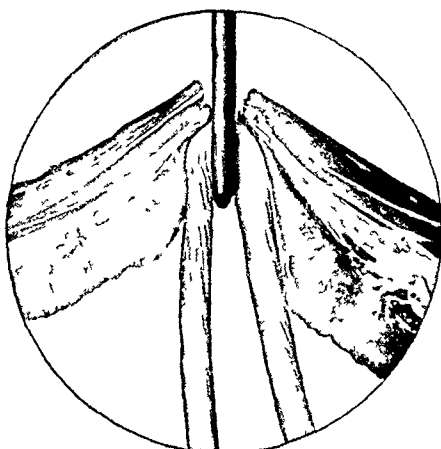
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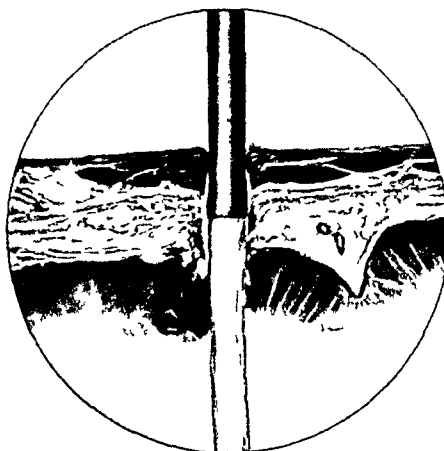
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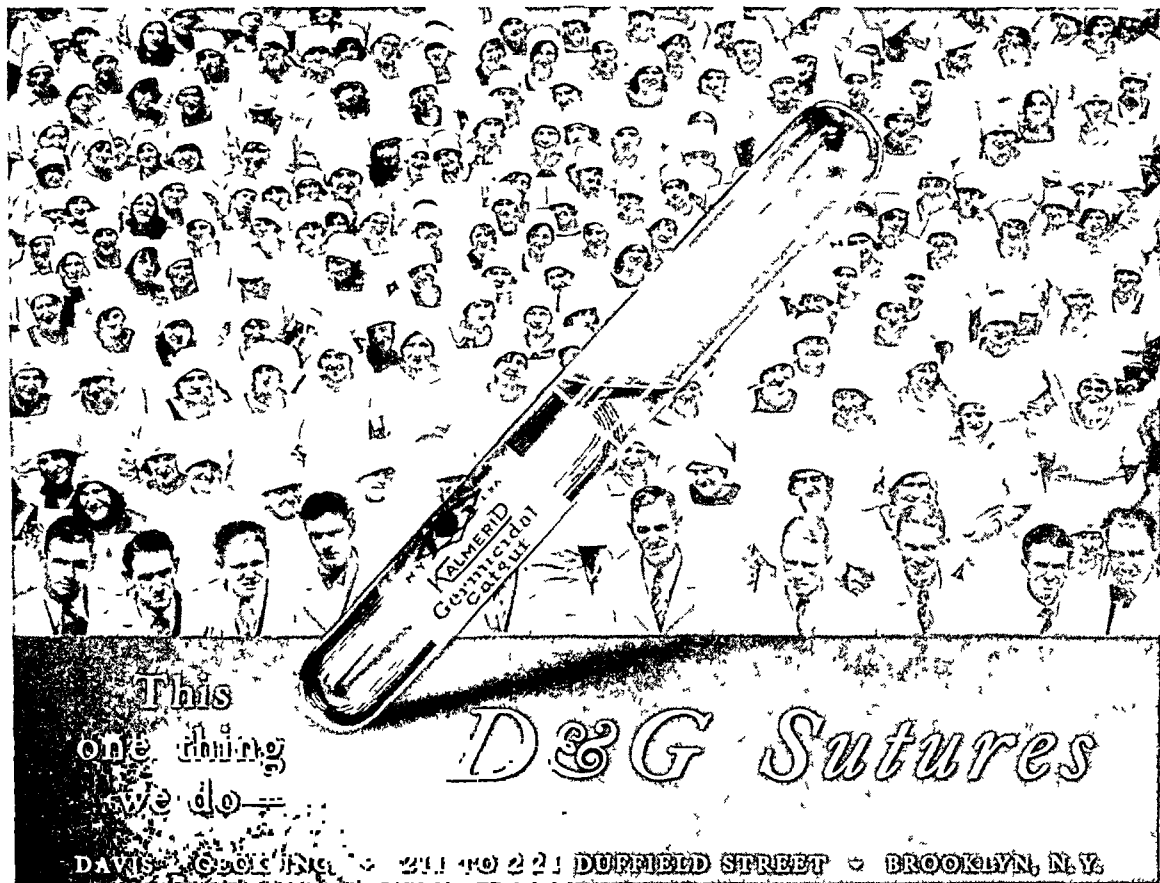
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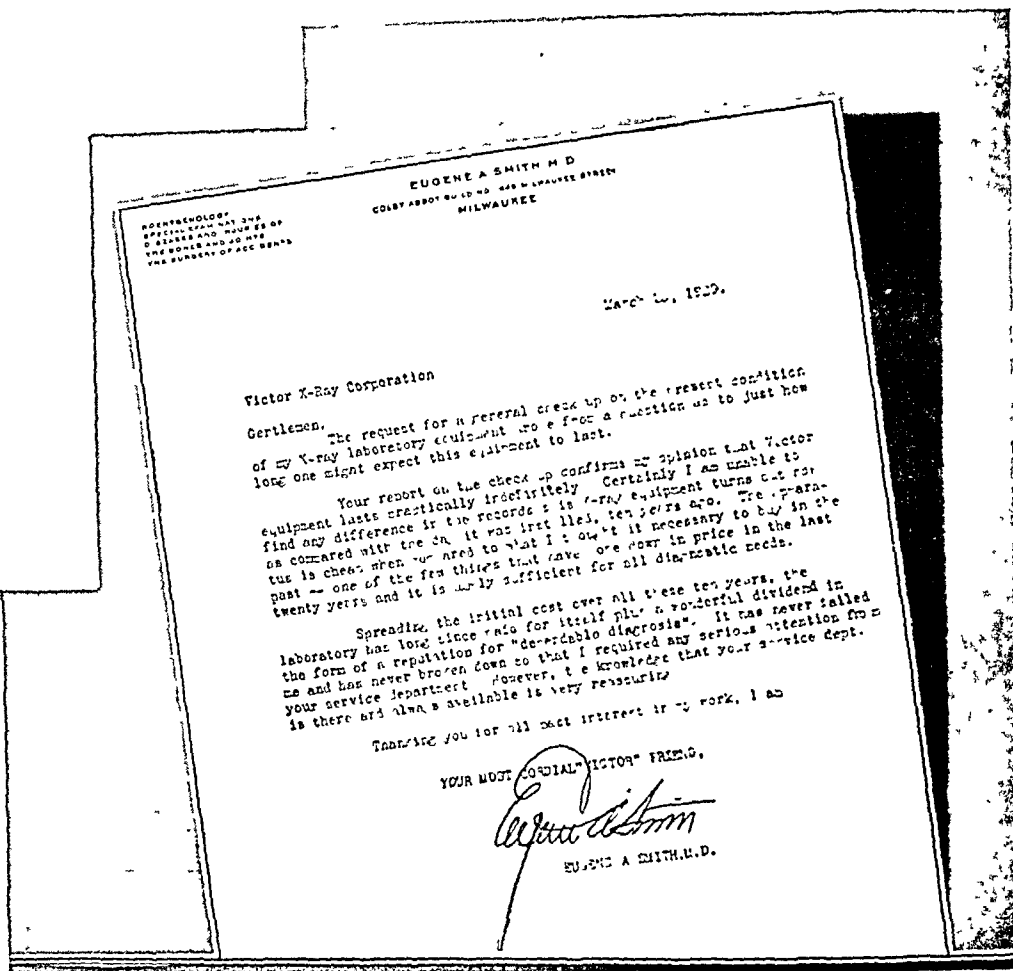
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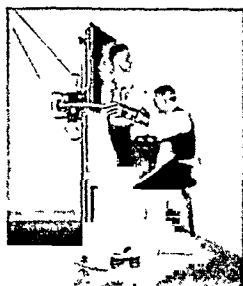
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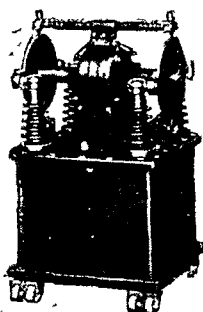
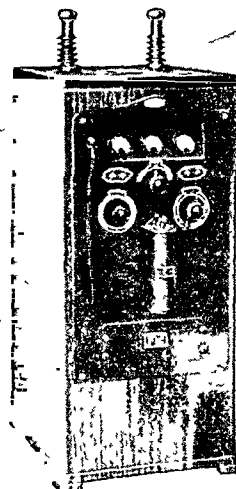
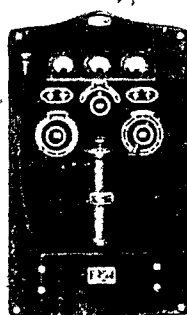
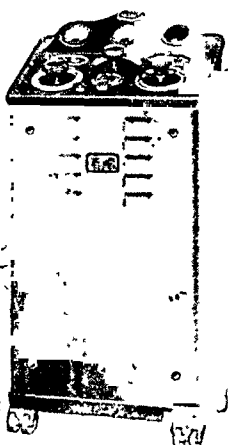
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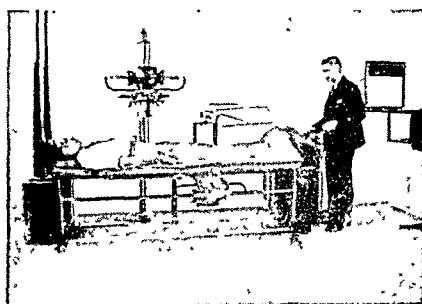
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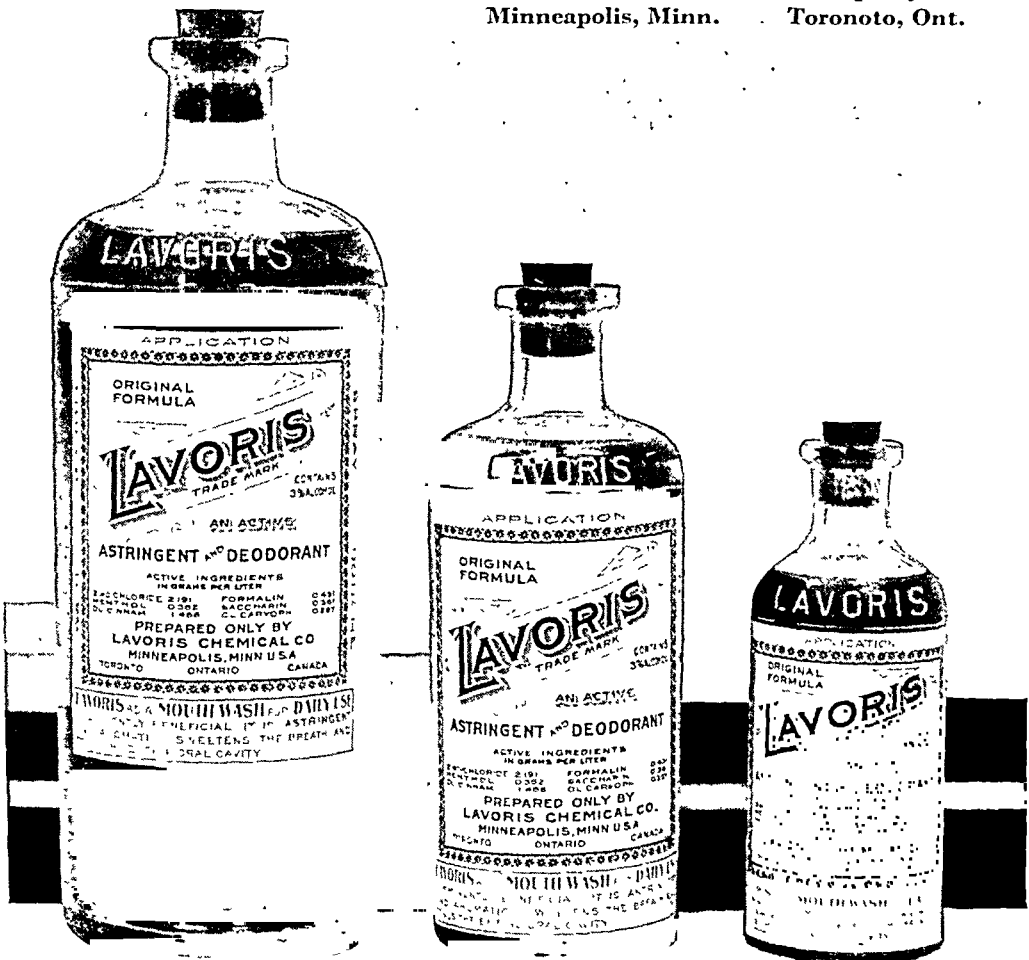
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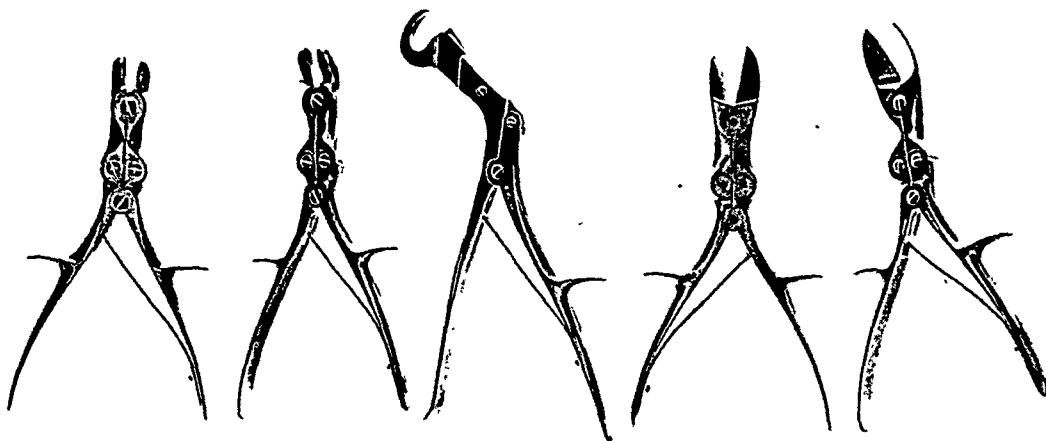
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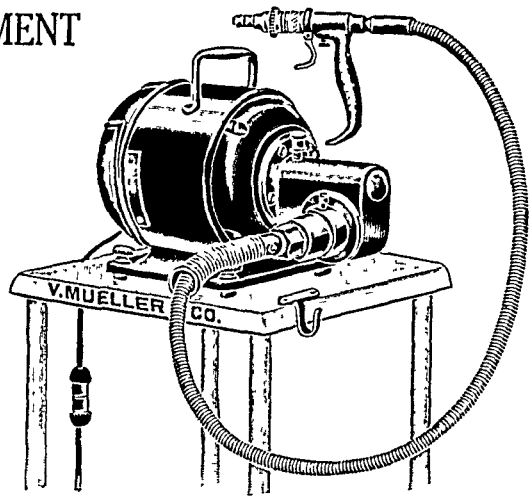
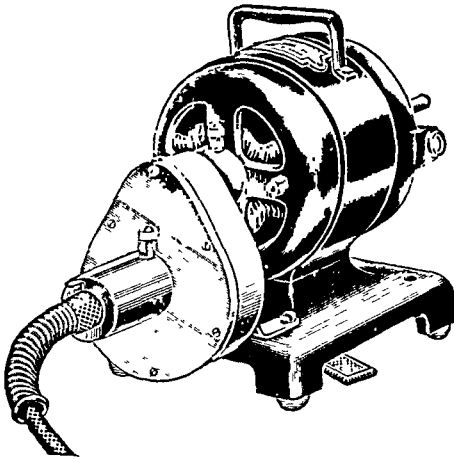
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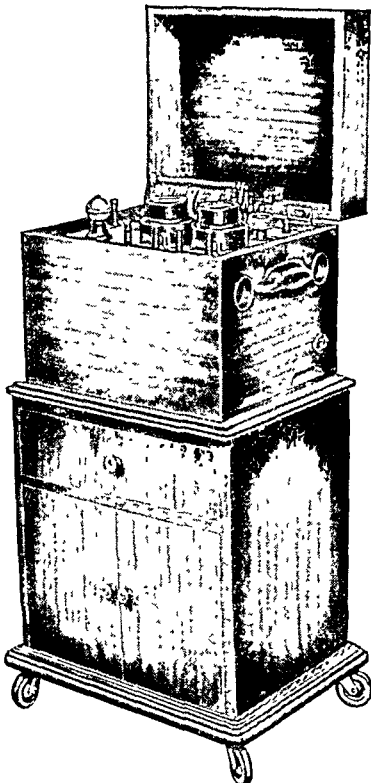
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THE NEWER CONCEPTION OF WOUND HEALING AS APPLIED TO PRACTICAL SURGERY*

ARTHUR E. HERTZLER, M.D., F.A.C.S.

HALSTEAD, KANSAS

THE old notion of primary wound healing is that the wound is agglutinated by a fibrinous exudate which in due time is resorbed and replaced by new fibrils formed by the fibroblasts. The new conception is that the wound is agglutinated by fibrin fibrils which remain to form adult fibrous tissue and are not replaced by the product of fibroblasts. That is to say, the transformation is due to chemical changes. This theory, advanced by me, has been abundantly confirmed by Baitsell and in large part by Werschinski.^{1,5,6} It is only when some condition exists which prevents the formation of fibrillar fibrin and a granular fibrin is deposited instead that it becomes necessary for the coagulum to be resorbed and be replaced by the product of fibroblasts. This is secondary healing. It is obvious therefore that the dividing line between primary and secondary healing in the new conception is microchemical. We may have secondary healing without infection. The confusion in wound healing is due to the fact that experimenters have dealt exclusively with infected material. It is this secondary healing caused by conditions unfavorable to the formation of fibrillar fibrin that is to receive discussion here. A secondary healing need not be infected.

We may state as an axiom that those conditions make for primary healing that

provide ideally for the formation of fibrillar fibrin in the coagulation of blood. It is the purpose now to see in how far this conception is borne out in the experience of every day practice.

As a preliminary it is advantageous to illustrate these different conditions by concrete examples. Union of loops of intestines are the most convenient material for study because it is easiest here to maintain asepsis.

When two loops of intestines are united by suture there is immediately thrown out an exudate at the angle of contact (Fig. 1). This exudate coagulates at once and may be said to be complete in 15 to 30 minutes. There results a bridging across of fibrils of fibrin. These fibrils are gradually converted into adult fibres of connective tissue without replacement. That these preliminary bundles are true fibrin is proved by microchemical studies. The proof that there is a gradual change into adult fibrin can be achieved only by following the process through many observations representing different stages. To secure these results it is necessary not only that the field of experiment be aseptic but that it be kept free from chemical substances which are inimical to blood coagulation.

If any factor enters which prevents the formation of fibrillar fibrin a granular

*Read before the Western Surgical Association, Chicago, Dec. 14, 1928.

fibrin fills in the space and agglutinates the surfaces (Fig. 2). This granular fibrin may be resorbed and be replaced by



FIG. 1. Angle between two coils of intestines is bridged by fibrillar fibrin which becomes adult fibrous tissue by process of chemical change.

fiber bundles deposited by fibroblasts. If the noxious agent is of a sufficient virulence it must be inhibited by the defensive forces of the body before the constructive agencies, the fibroblasts, can become operative according to the generally accepted teaching as applied to the usual injuries to the soft parts. In certain situations the purposive factors in pathology come into play and instead of the granular fibrin being absorbed and replaced by fibrous tissue as in, say, infected skin wounds, the formation of adult fibers does not follow and the previously agglutinated surfaces are released. This is a common observation when intraperitoneal abscesses are drained. The encapsulating adhesions are released and after the course of months no trace of the adhesions remain. I have been able to follow this in my window experiments.³

The surgeon should know that his wound may be absolutely free from bacterial infection and yet the wound really does not heal by primary intention. There are many conditions aside from infections which prevent primary healing, in whole or in part. A firm grasp of this conception is, in my opinion, the very foundation of correct surgical technique.

I propose now to detail some of the non-bacterial conditions that prevent primary healing.

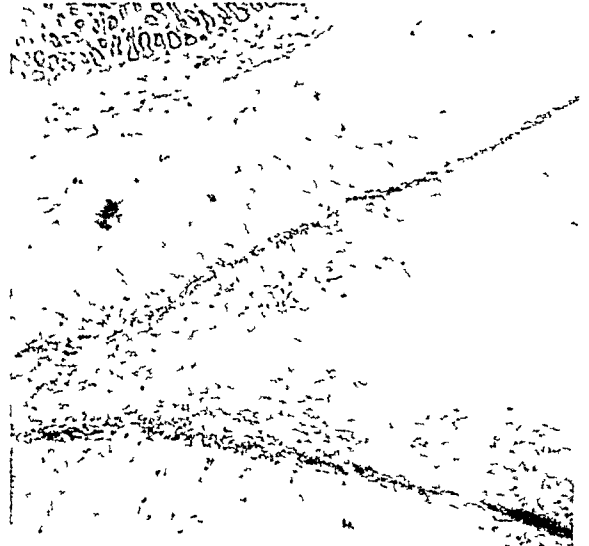


FIG. 2. Angle between two intestinal walls filled by granular fibrin which, as the inflammatory reaction subsides, is resorbed and intestinal coils are freed.

INTERPOSITION OF FAT

Perhaps the most common cause of failure of wound healing is the intervention of fatty tissue. Fat, as we have learned in vessel anastomosis, prevents the coagulation of blood, therefore prevents the formation of fibrillar fibrin. We must therefore free surfaces from all fat if we desire prompt healing. This applies particularly in closing abdominal wounds, particularly wounds to which stress will be applied during the process of healing, notably primary and secondary hernias. This fact applies with particular force in intestinal anastomosis in areas where the gut is in part covered with fat. Fat uncovered by peritoneum will not heal and we must remove it from the surfaces we expect to unite. If we cannot secure peritoneal surface for our opposing surfaces our next best material is muscle wall. In using muscle wall, as will be set forth more fully in the discussions in the repair of hernia, we must know that we are not dependent on the healing of muscle for muscle itself

does not heal, but on the production of a fibrosing myositis; quite a different process than the healing of opposed peritoneal surfaces.

Normal fat may be prevented from interfering with wound healing by the mere mechanical process of keeping it out of the way. By exposing the fascial surfaces we desire to heal we produce conditions conducive to ideal wound healing. Necrotic fat infiltrates and invades the suture line even when correctly coapted. This fat necrosis is produced by injury as when included in a fascial suture and is an aseptic necrosis just as we see it in the omentum in pancreatic disease. We can produce it experimentally by depriving fat of its nutrient supply and as we see it commonly in blunt injuries, particularly in blunt injuries of the breast. Necrotic fat is produced in the course of operations by ligating masses of fat with the vessels and by rough handling, especially by retractors. Fat, therefore, must not only be got away from the line of injury but it must be so handled that it shall not be traumatized.

ACTION OF NECROTIC SUBSTANCES

Other aseptic necrotic tissue acts in the same way; hemorrhages into tissue whether operative, traumatic or pathological, as hemorrhage into cysts. The common source of traumatic necrotic tissue is the result of bungling and rough operating, particularly the use of retractors. The common form of the trauma is due to hemorrhages, extrauterine pregnancy, blunt injuries and the like, occlusion of vessels from any cause followed by exudates into the tissues as one sees commonly in the intestinal walls from occlusion from any cause. Often have we complicated the scene by adding operative traumatic exudation. Pathological hemorrhages we commonly see in hemorrhages into cysts. Such blood, as it undergoes changes after it has escaped, is aseptic but it prevents the coagulation of blood with which it comes into contact, therefore prevents

wound healing. The old surgeons knew that postoperative hernias were particularly apt to follow any condition in which escaped blood had to be conducted out of the wound, particularly hemorrhagic cysts of the ovary.

DIGESTIVE FERMENTS

Digestive ferments act in like manner. This must be remembered in operating in regions where such fluids are apt to enter the wound. This applies to the salivary regions and in the gut tract. We know by experience that the upper gut tract is more likely to make trouble than the lower even though the lower is much richer in its bacterial flora. In the lower gut tract, however, conditions are complicated by the presence of fat, either normal or traumatized because the large gut is in part extraperitoneal. In operating in regions where digestive fluids are unavoidable the best we can do is to conduct these fluids away from the site of our wound. We have learned well enough the necessity of doing this in the urinary tract for urine prevents the formation of fibrin through less actively than the digestive fluids.

CONSTITUTIONAL CONDITIONS

Any constitutional condition which alters the coagulability of the blood prevents wound healing. Mere slowness of coagulation time is not the only factor, for the time may be prolonged and yet perfectly formed fibrillary fibrin be produced. On the other hand the time may be normal but the fibrin be imperfect. Those conditions likely to be followed by secondary hemorrhage are particularly likely to be attended by imperfect wound healing, obviously for the same reason.

ACTION OF TISSUE EXUDATES

This brings us up to the dreaded complications of postoperative thrombosis and embolism. These are due generally to the formation of aseptic thrombi of granular fibrin. The fundamental change is in

the edematization of tissue in which the vessel lies. This edema permeates the vessel walls and so changes the sub-endothelial substance which forms the fibrin but is unable to produce a fibrillar fibrin. Of course septic thrombi may act in like manner but we are not concerned now with septic conditions. Clots are formed but lacking fibrillar fibrin they do not organize and by normal pressures or manipulations are detached from their imperfect anchorage to the vessel wall and are sent on their way of destruction. It is the same process which takes place when endocardial vegetations are detached, except these are usually septic in origin and frequently contain living organisms. The vagaries of postoperative thrombosis and embolism of course transcend our knowledge. No one before or since John Hunter has visioned the fundamental factors. Autopsy studies have muddled rather than cleared the waters. I believe it is safe to say Hunter knew better than any one the relation of reactive process to the circulation and his work still is the latest, in fact the only work on the subject. The only practical point we can deduce from the study of the subject is not to ligate vessels in tissues which are in a state of mild reaction. This is particularly true in tissue that is edematous. Either leave the lesion alone or ligate proximal to the irritated area. In the omentum, for instance, the ligation must be made central to any diseased tissue.

THE FORMATION OF ADHESIONS

The science of wound healing finds its most fascinating application in the problem of the formation of adhesions.

The question of what constitutes an adhesion and what a normal or aberrant fold has complicated the pathology of the abdomen from the beginning.

The only foundation for the study of adhesions is a first hand knowledge of normal anatomy including the early developmental stages. Unless one knows what normally happens it is impossible to

properly appreciate the abnormal. It is profitable to study briefly the genesis of unusual folds. The common site of the folds is the subhepatic and ileocecal folds. In the embryo, as the organs take their places, it is common to find extensive folds which in the adult would be aberrant folds. These are most pronounced about the gall bladder and duodenum. It is common to find the gall bladder with a complete mesentery. The gastrohepatic fold likewise is often very prominent. They are much more common in the embryo than in the newborn. The failure to recognize these folds as normal variations in many instances causes the operator to fail to find the real lesion because he feels that in these folds he has found the real cause of the patient's complaints.

The ileocecal region is an even more interesting field for the study of aberrant folds. The inconstancy of the position of the appendix has often been the subject of fable and song. That this diversity of position is due to the variation in the mesoappendix and the manner and degree of rotation is obvious. Yet such variations are commonly cited as evidence of "chronic" or past inflammation. The degree of vascularity of the peritoneum has much to do with the prominence of these various folds.

How shall one, gut in hand, know whether he has an adhesion or a normal or aberrant fold? First of all by a thorough knowledge of the range of the normal. The gut with its questionable fold must be studied in situ before any traction has been made on it. By traction one can make a "fold" in almost any direction. The ridiculous fallacy of the Lane "kink" was possible only by wholly disregarding this very obvious fundamental fact. If a band does not interfere with the function of a gut when the gut is in its normal position it probably is not an adhesion and most certainly it cannot be hypothecated as the source of symptoms. A fold cannot be regarded as an adhesion unless it could

not have been formed by any aberration of normal genesis. In rare instances one cannot be sure.

The second factor in the diagnosis of adhesions is a full appreciation of the fact that adhesions are not the result of infective processes. Adhesions due to and at the site of infection are protective and temporary and are released after the process has run its course. When permanent adhesions follow infections they occur adjacent to the main pathological lesion and not in its immediate site. The residual adhesions, for instance, following an acute suppurative appendicitis are to be sought not in the immediate vicinity of the appendix. They occur in a region adjacent to an infective process in which the irritation is just sufficient to produce an exudate capable of forming a fibrillar exudate. Adhesions following appendicitis are to be sought therefore not about the appendix but in the pelvis or over the ascending colon or somewhere in the great omentum. Alleged adhesions about the appendix are almost sure to be folds unusual in the experience of that particular operator. It is common to mistake in these situations normal folds made more prominent by hyperemia or subperitoneal edema.

The ephemeral nature of adhesions about an infection is perfectly clear to those of us whose experience extends back twenty-five years or more. It was the common practice then simply to drain the abscess and often a lapse of from three to six months occurred before reoperating for the purpose of removing the appendix in order to prevent a subsequent attack. We know how regularly the appendiceal region had been wholly freed from the walling-off process which prevented the spread of the infection.

The formation of permanent adhesions by aseptic irritation is well illustrated by a simple experiment. If the transversalis fascia is exposed, without opening the peritoneal cavity, and a pledget of gauze is placed against it and the wound closed the great omentum will migrate to this

point and form a permanent adhesion (Fig. 3). The irritation of the gauze causes an exudate on the peritoneal



FIG. 3. Omentum adherent to abdominal wall, result of aseptic peritoneal irritation produced by placing pledget of gauze over parietal peritoneum and under abdominal muscles without opening into abdominal cavity.

surface which attracts the great omentum. In a very similar manner the omentum seeks the line of suture of an abdominal wound if an irritation is produced or a surface not covered by endothelium is allowed to remain.

MUSCLE-FASCIA HEALING IN HERNIA OPERATIONS

Whether or not muscle can be made to heal to Poupart's ligament in the repair of inguinal hernia has recently been the subject of a lively argument. That the conjoined tendon does heal to the ligament is of course common knowledge to those who have had to reoperate on hernias operated on by Bassini's method.

As was noted above, muscle fibers themselves do not heal. Healing occurs only by virtue of the connective tissue of the muscle. In instances where this is not sufficient one must so operate as to create fibrous tissue. Dupuytren's contracture is a fibrosing myositis the result of trauma from the injury or from too tight bandaging. We need such fibrous tissue in the repair of inguinal hernias

and we procure it by partly strangling the muscle. Here, however, we do it with sutures. By tightly sewing the conjoined

importance but it should not be absorbed before five or seven days. Sutures obtained from the patient's own fascia, either in



FIG. 4. Myositis produced by sewing muscle to fascia. Muscle fibers distinguishable at left. In center new fiber bundles are forming.

tendon to Poupart's ligament we produce a fibrosing myositis; the newly produced fibrous tissue forms a real fibrous union with the ligament. This is true provided, of course, some error in technique does not prevent it, such as infection or the interposition of fat. If the sutures uniting the muscle to the tendon are not drawn tight enough to thus injure the muscle union will not take place. The time required for healing varies from twelve to fifteen days. Figure 4 shows the degree of edema and round celled infiltration which results after four days. After healing is completed and the round celled infiltration has subsided heavy fascial bands prevade the muscle for some distance about the line of suture (Fig. 5). The degree of tension on the suture required to produce the desired muscle injury is about as much as one would use in ligating a vessel of considerable size. That is about all that one can comfortably apply with the unaided fingers. The same principle applies to the repair of postoperative hernias where muscle to muscle or muscle to fascia are united. It goes without saying that in muscle containing but little fibrous tissue the tension on the suture must be limited to what the muscle will stand without cutting through.

The kind of suture used is of little



FIG. 5. Connective tissue bundles between muscle fibers much increased. Specimen taken from conjoined tendon 2 cm. from Poupart's ligament four years after Bassini operation. Fibrous union between muscle and ligament had taken place.

the immediate site of the wound or elsewhere, as from the fascia lata, has nothing to commend it aside from saving the price of a tube of catgut. The fibrosing myositis is the essential thing. The agent used is immaterial.

THE QUESTION OF DRAINAGE

Surgical drainage is done for the purpose of conducting to the surface fluids that have collected in natural or artificial cavities, or to furnish means of escape of fluids we fear will accumulate in the tissues and cause trouble. In the first we do not desire primary healing of the wound and in the second we do; we must conduct ourselves accordingly.

In the first instance we desire the wound

kept open until the diseased condition can heal. We may cite as examples the drainage of an infected gall bladder or a pelvic

tubing. Such elation is just as wise as that of the old surgeons pointing to an abundant production of free pus.

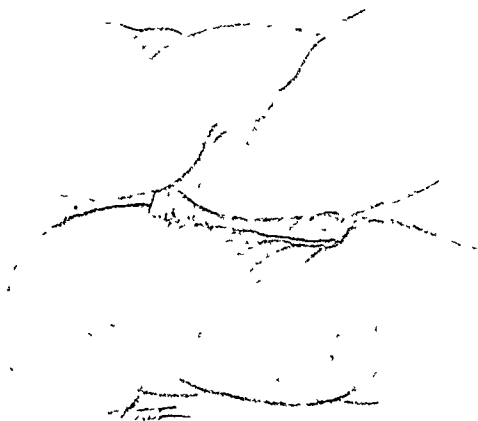


FIG. 6. Pledget of gauze placed between two intestinal coils. Fibrin covers in gauze at left. At right fibrin bundles were torn by manipulation.



FIG. 7. Piece of rubber tubing lies between loop of intestine. Fibrin piled up about rubber but nowhere does it cover edge.

abscess. We want to use a material which is unfriendly to the closure of the wound or in other words a material which will inhibit the coagulation of the wound exudate. Rubber is commonly used for this purpose and it meets all scientific requirements.

When we drain a clean wound we do so to avoid the accumulation of exudate. Here we need a material for drainage purposes which will favor coagulation, hence lessen the amount of fluid exuded and by so much lessen the amount to be conducted out of the wound. Here obviously rubber is wholly unsuited for the very reason that it was desirable in the first type of drainage. Coagulation is expedited in escaped blood by the presence of a foreign body. The exudate which flows into a fresh wound is in part blood, in part tissue fluids but the laws of coagulation apply to both. The best foreign body easily available to excite coagulation is a gauze wick. This aids the coagulation of the escaped fluids and by that degree lessens the amount to be conducted away. Some surgeons argue in favor of the use of rubber because they find more fluid has been conducted away by the rubber

A simple experiment is easily performed which shows the difference of reaction of the tissue to rubber and to gauze. Gauze is quickly covered by coagulated exudate (Fig. 6) while rubber is not (Fig. 7). More elaborate experiments such as the use of rubber tubes and gauze strips show the difference more plainly.

There is still another side to the problem. We apply ligatures to cut vessels to control hemorrhages. This is a temporary measure only. We expect fibrin to form about and within the vessels which in time develops into fibrous tissue and thus the severed vessel is permanently sealed. Now the presence of gauze in the neighborhood of the ligated vessels aids the production of fibrin, hence favors the healing about the ends of the vessels. Rubber, on the other hand, promotes the secretion about the tissues and by so much discourages the healing of the wound. If the wound is not healed by the time the ligature is absorbed secondary hemorrhage ensues. Therefore rubber should never be used in a wound that one desires to have heal by first intention and in which secondary hemorrhage is feared.

ON THE TECHNIQUE OF SKIN GRAFTING

The grafting of skin is the healing of epidermis or dermis taken from one place

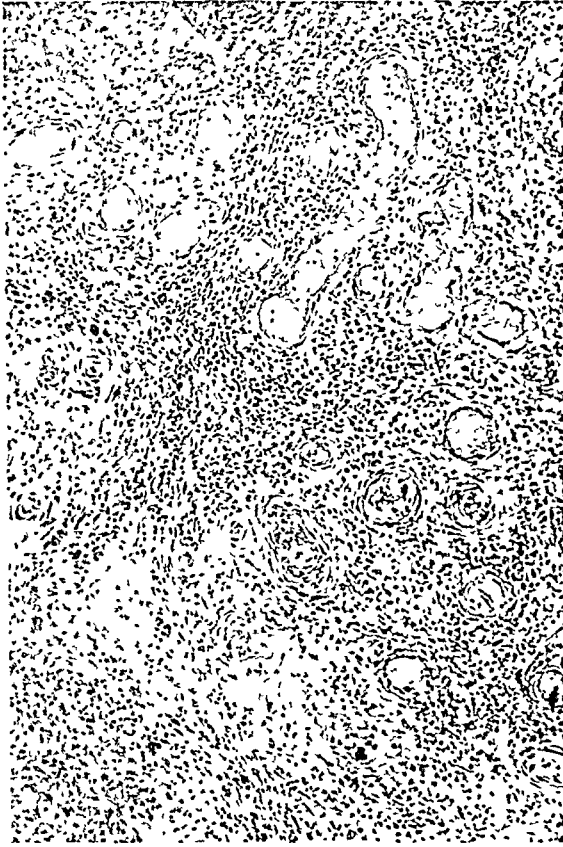


FIG. 8. Granulation tissue four weeks old. Numerous vessels with thickened walls are visible. Connective tissue is palely staining.

and applied to another. It is, therefore, purely an experiment in wound healing. The point to be attained is to remove the graft to its bed before the serum has time to coagulate, so that the liquid exudate of the graft and that of the bed may coagulate simultaneously and thus form fibrin bridges from the one to the other. This requires a reasonable promptness and requires that the bed be prepared before the graft is removed. The old practice of floating the graft in saline solution was the worse practice possible because this tended to remove the serum whose coagulation produces the attachment.

The question of the preparation of the bed requires a word. Fresh wounds are desired because they are presumably free from infection. Granulating surfaces must

be accepted in many cases. If non-infected they are almost ideal. If the granulations are young, excoriation with a piece of gauze is sufficient to produce an exudate competent to take the graft. There are two conditions which may cause a failure of the graft: (1) infection which prevents the formation of fibrin; (2) an unsuitable bed to take the graft. When granulations have attained a certain age, placed in a general way at three or four weeks, the vessels of the granular area undergo endovascular changes (Fig. 8) which makes it impossible to secure a suitable serum in which to imbed the grafts. In such cases the granulation tissue must be removed by means of a knife or curet in order to secure a fresh surface. Of course the bed must be smooth so that the graft can lie evenly and touch the bed at all points.

CONCLUSIONS

Primary wound healing occurs by the coagulation of exudate forming a fibrillar fibrin which is converted into adult connective tissue by a chemical process.

Wound healing is prevented when the exudate forms a granular fibrin. This may be due to infection or to the presence of some chemical inimical to the formation of fibrillar fibrin.

Healing of muscle to fascia is dependent on the production of a myositis which results in the production of fibrous tissue.

Secondary hemorrhage and emboli result when the vessel wall is so irritated by chemical means that a fibrosing thrombus is not formed.

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ETIOLOGY OF GOITER*

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COLORADO SPRINGS

THE theories advanced as to the cause of goiter are many; these theories have been given much study by many authorities on the subject of thyroid disease. Conclusions based on facts are few, yet from such studies we are able, with much precision, to establish some truths that are not questioned.

The need of a more united opinion prompts a review of the conclusions based on the studies of all coworkers in this particular field, namely Etiology. A careful study of the literature as put forth in this contribution will be a collaboration from this survey.

Alfred Stille, 1871, in an address said: "Science itself is unstable. The science of the last century is the folly of today, and much of that on which we pride ourselves as certain will be found in the lumber room of the next generation." This is certainly most true as it pertains to present-day studies of the thyroid gland.

Various etiological factors have been proposed, elaborated and scientifically investigated by many writers, yet when the question is put, "What are the causes of goiter?" few indeed will reply with a positive answer.

In discussing the etiology of goiter, we shall not take into consideration carcinoma, sarcoma, syphilis or tuberculosis, since they occur not only in the normal gland but also in the goiterous thyroid and when found in the thyroid should be classed under the head of diseases of the thyroid gland and not as factors in the production of goiter. Since malignancy is more common in goiterous gland than in the normal gland, the deduction should be that goiter is a factor in the occurrence of malignancy rather than malignancy producing goiter. To substantiate this premise, Sarbach states that "Carcinoma, sarcoma and

syphilis do not produce any significant changes in the thyroid."

Many consider psychic trauma a contributing factor. A careful study of a series of cases and deductions made from a review of the literature would seem to indicate that psychic trauma can be best explained on the basis that the manifestations of the disease are brought to our attention, rather than being a factor in its etiology. Consequently in an individual developing manifestations of toxic goiter, following psychic trauma, one should look for a preexisting goiter.

Some writers lay much stress on sex imbalance as an etiological factor in exophthalmic goiter. I have been unable to see any preponderance of goiter in unsatisfactory marriages over married life under most favorable conditions. I do think that goiter *per se* has a tendency to develop incompatibility, and often many of us have observed patients, cured of exophthalmic goiter, who see life in a new way from every standpoint. Such can also be said of nervous and physical strain.

Many recent opinions call attention to heredity as a factor. Morrison mentions three family groups affected with exophthalmic goiter. Heredity stands out as an important factor in the cause of goiter. We have not given this phase of the subject sufficient scientific study and experimental consideration. Much work has been done in endemic regions and localities where goiter has been present for generations. We think this a great mistake. Such investigations should be carried out in new countries where goiter-producing families can be studied. It is for this reason that the Rocky Mountain States offer one of the best fields of study.

Experimental work by many investigators shows conclusively that goiter can

* Read at the Annual Meeting of the American Association for the Study of Goiter, Dayton, March 25-27, 1929.

be produced in the offspring of animals; that is, normal and goitrous offspring can be produced at will. Such results have been carried out on pigs and dogs with convincing results.

Hardly can an investigator now be mentioned who does not believe that familial influence is a factor. I have a large group of families in which we have been able to study the heredity feature in three generations in Colorado Springs, the original member of each family developing goiter while living in other sections of the states.

Many investigators are positive that goiter is not often inherited, and consider family history as unimportant; others after careful study of many cases are convinced that heredity is a prominent factor. So-called goiter districts are on the increase in the United States. It is quite evident that only three generations are necessary to develop a goitrous focus. There must be some reason why daughters of goitrous mothers develop thyroid disease. Certainly a higher per cent of girls than boys become victims of abnormal thyroid.

No one should question the maternal influence as an etiological factor notwithstanding the fact that no absolute explanation of how it takes place has been put forth. Is it a difference of thyroxin needs of different individuals or the inability of the mother to produce sufficient thyroxin to meet the requirements of the fetus, or is it direct transmission of some infection to the offspring after birth in the children who do not suffer from congenital goiter? Surely there must be some reason why, if a mother has goiter, many of her girls will develop the disease and virtually all the girls and a good number of boys in the third generation will become affected. A most interesting fact is that the family need not remain in the locality where the original member developed the disease. They can be removed to another community and the peculiar phenomenon remains unchanged. It is further interesting that

no other family in the same block or locality will develop the disease in the new community, while goitrous grandmothers have many goitrous offspring, although buying groceries from the same store and drinking from the same water supply as their more fortunate neighbors.

Much experimental work has been done with diet in the production of goiter. McCarrison has probably devoted more time and carried out a more thorough and extensive study than any other investigator. He believes that a particular type of goiter can be produced by a diet deficient in manganese, insufficient in vitamins in all classes, and insufficient in roughage. This particular type of goiter develops despite the adequate ingestion with the diets of iodine in the form of potassium iodide. He considers that there is no organ in the body, with the exception of the adrenal gland, which is more sensitive to faulty food, and none whose structure is more prone to variations as a consequence of different food faults, than the thyroid gland. Food contaminated with fecal bacteria can unquestionably produce goiter and goiter can be prevented by increasing the intake of iodine proportionally to the unhygienic conditions. Many investigators confirm this opinion, but do not agree that the food or bacteria is the noxa producing agent, but claim lack of iodine is the basic factor.

Drinking water plays a most important part in the etiology of goiter. Certain kinds of water will produce experimental goiter. Drinking water, as a cause, is one of the oldest and best supported theories by fact and experiment. There is much difference of opinion as to what part the water plays in the production of goiter. Is it a lack of chemical agents in the water or bacteria contained therein? The theory which incriminates the deficiency of iodine is concurred in by a large group of investigators; some believe in the infectious germ in the water, while others combine the two theories.

Messerli showed that drinking water in

regions where goiter was endemic practically always came from shallow springs and was always infected. He produced goiter in white rats by causing them to drink water from a part of Switzerland in which goiter was endemic. He also produced goiter in rats by giving them water that had been boiled, and passed over human feces. I not only mention his work to support the theory that water is a factor, but also to prove that infection is the active agent in his experimental work.

Disturbed internal metabolism has been taken into consideration as a cause of goiter by some investigators. They contend that normal blood possesses considerable antithyroidal power, and give as proof the statement that the blood of Graves' disease patients acts on the metamorphosis of the tadpole in the same way as thyroidal substance. They seem to think that the blood of Graves' disease patients must have lost its peculiar antithyroidal power. This, of course, applies to the so-called Graves' disease or hyperplastic goiter.

Is lack of iodine a real cause of goiter, or is it only a factor? Certainly its use in an experimental way has shown that its administration will prevent goiter in the offspring of animals, fish, etc. Does it supply a deficiency in the individual and thus prevent the disease or does it prohibit the invasion of bacteria by keeping up the normal iodine content of the blood? McCarrison's work is most interesting. Water supply in one section may be sufficient in iodine, and yet goiter may be endemic therein, while the water supply in another section may contain no determinable amount of iodine and yet goiter may not be endemic therein. Drinking water containing a large amount of iodine (300:1,000,000) does not prevent the occurrence of the disease in the presence of a high degree of bacterial impurity. This proof was obtained by actual experimentation on man. His further investigations do not show high incidence in individuals, on a diet poor in iodine; on the contrary,

the rice-eating Indians are rarely affected with goiter.

How does lack of iodine produce goiter? Is it a deficiency in all the anatomical structures in the body, or is it a real need of the thyroid gland, or is it a lowering of the threshold of the iodine content of the blood below normal, permitting infection to step in and attack the thyroid gland? The latter seems quite possible if due consideration is given to all opinions as expressed by the many scientific investigators in this particular field.

Iodine in the blood has an antiseptic value, it also has germicidal and vermifugal value in the bowel as demonstrated by Chandler.¹

The experimental work of Green and Mellanby with diets deficient in vitamins seems to prove that when rats are fed on this particular diet they live for a while, grow weak and die. Ninety-three rats were used for the experiment while 50 controls were given a supplemental diet. All but 20 of the 93 rats, at postmortem examination, had infections in one or more parts of the body while none of the controls were so affected. Conclusions from this work would lead one to think that lack of vitamins is not the direct cause of goiter but is only a factor and that after all infection may be the basic cause. The same reasoning can be applied to the oft-repeated opinion: "Soldiers developed goiter in the trenches due to psychic trauma." Here again we find infection a consideration. I am sure there was never a greater source of infection than that found in the trenches. So to me, infection should take first place as the probable cause of goiter, originating from this source, in place of psychic trauma.

The studies of Cole, Womach and Gray show that 74 per cent dying from diseases other than that referable to the thyroid had definite damage to the gland, indicating that the gland had suffered many attacks from infection and while definite goiter had not become manifest, the

¹ *Quart. Bull. Mich. Agric. Coll.*, (1924) p. 24.

pathology was quite characteristic. Experimental work by these observers show much the same lesions in animals dying from induced infections, claiming that organisms inhabiting the intestinal tract are more prone to produce such changes in the thyroid, than the pyogenic group.

Does a thyroid recover from an infection and remain well, showing only evidence (enlargement, etc.) as does any other organ, and is a permanent cure obtained unless an infecting agent makes a second attack and a new storm takes place in the gland?

The function of the thyroid is iodine metabolism; it is quite natural then, that the iodine-content ratios should change when the gland is disturbed by infection; consequently the opinion expressed that lack of iodine is the cause of the disturbance may be paradoxical.

Helwig in the discussion of the improvement in a patient after the administration of iodine (Plummer method) states that he doubts that the change in the amounts and quality of the colloid (which follow the Plummer treatment) explains the clinical improvement completely.

Iodine administered to goiterous patients, suffering from thyroid disease, may antagonize the infections, neutralize the toxins, and benefit in this way, rather than supply a deficiency to the gland.

If infection is the sole cause and all other factors secondary then the many physiological and pathological phases observed may be the results of a thyroid gone mad from stimuli of various toxins of bacteriological origin.

Summing up the world's literature, the causes of goiter may be placed under the following heads: Infection, lack of iodine, psychic trauma, sex imbalance. Factors influencing etiology may be mentioned as follows: Food, water supply, heredity. Most authorities agree that infection is the cause of endemic goiter. Many authorities think infection to be the cause of exophthalmic goiter. Many think that lack of vitamins in white bread is the cause of the hyperplastic type, as seen in the

white-flour eating people. This type is toxic, shows early heart signs, and is very common in England and the United States. Food is a factor in infection and lack of iodine. Water supply is a factor in infection and lack of iodine. Heredity is a factor in infection, psychic trauma, and sex imbalance. Lack of iodine many consider only as a factor in influencing infection. I quote a comparison made by Vallerio: "Quinine cures malaria, quinine prevents malaria; lack of quinine does not cause malaria. Iodine prevents goiter, cures some types of goiter, but lack of iodine never causes goiter." This opinion is concurred in by many European authorities.

Prophylaxis is affected by improving the water and food supply, and the administration of iodine; thus strongly supporting the theory that infection and lack of iodine will solve the question of etiology. And the solution is up to our country, as suggested by Blum who states, "Could not the U. S. A., which has every possibility to carry out experiments on a large scale, make an effort to eliminate the cause of endemical "Struma ad cretinismum vergens" by consistently purifying the drinking water (filtration)?"

In the second place, the "Struma ad morbum Basedowii vergens," should be examined by chemical and biological blood tests in its relation to the organism in general and, according to the results achieved, should be combatted by intense doses of antithyroidal blood.

OPINIONS OF INVESTIGATORS

KESSEL AND HYMAN: Etiology: 1. Predisposing Causes. The factors that predispose to this syndrome are at present unknown. Manifestations of the disturbance have been present in most of these patients as far back as they can remember. Whether the predisposition is inherited or whether it is due to some early environmental factor remains to be decided. 2. Exciting causes. As exciting causes may be mentioned (a) sex epochs; (b) focal infection; (c) psychic insult.

ASCHOFF: I myself am of the opinion that goiter can have many different causes. To differentiate the physiological swellings of the

thyroid in the newborn, in puberty, and in pregnancy, which must be laid back to endogen causes, from the endemic goitric swelling, which makes it imperative that the weight of the thyroid during the whole life is increased; such a swollen goitric thyroid shows also the swellings of the newborn, of puberty, and of pregnancy, so that here are summed endogen and exogen factors.

Now what concerns the exogen factors of the endemic goiter swelling, the mixture of water, air and food plays a great roll. I am of the opinion that the relative lack of iodine and the relative richness of lime in the locality of the individual will be a furtherance to the growth of the endemic goiter. To that, must also be added the changing individual disposition which is present in each individual characteristic (matter changing form). Further, all those outside factors, which influence the ground-work, also play a roll in the commencement of endemic goiter. So, before everything else, the climate. It is known that in the whole world certain heights of the mountains will favor the growth of the endemic goiter. Here strong gatherings of fog, sparse sunrays, cooling-off, impregnating moisture, etc., will play a certain roll. The writings of Tanabe, Schmitz-Mohrmann, Sorour, worked out at my institute will let the importance of the different factors be clearly recognized. Just lately have we discovered that darkness is an important irritating factor for the thyroid. In the same direction will also work the cooling-off. (Cramer-London.) In opposition to that, light and warmth will act as a quiescent on the thyroid.

So in the foregoing I have named some of the most essential factors, that to my mind, are to be considered in the endemic goiter growth. However, I must emphatically state that the Basedow thyroid and the Basedow impregnated goiter are something entirely different. Here conditions are similarly like puberty swelling and pregnancy swelling; an endogen produced irritation of the thyroid, which can affect a normal thyroid as well as an endemic goitric thyroid.

DECOURCY: Fatigue, in my mind therefore, accounts for all the manifestations of goiter, and agrees in part with other theories, only explaining them differently, focal infections, lessened iodine, excessive proteids, adolescence, pregnancy, atmospheric changes, climatic and material differences, facts which we all recog-

nize as of casual relationship to goiter, act by lessening the normal zone of endurance and initiate an overactivity of the thyroid gland, when this limit or threshold of endurance is exceeded.

GALLI-VALERIO: 1. The causation of endemic goiter (goiter, cretinism and deafmutism) is not yet fully understood.

2. Of the numerous theories, that which regards drinking water as the cause of goiter is at once the oldest and best supported by fact and experiment.

(a) Detection of contamination of the drinking water in all endemic districts.

(b) Abatement and disappearance of the endemic under improved conditions of supply of drinking water.

(c) Appearance of goiter in animals which have been watered from sources in endemic districts or with artificially contaminated water.

3. The noxa of goiter in drinking water is either a specific substance or a specific germ or group of germs, especially of the intestinal flora, which produce toxic substances that act upon the thyroid gland. By consequence, disinfection of the bowel is beneficial, and a regime favoring constipation the reverse.

4. The drinking water theory does not exclude the possibility of other vehicles for spreading the endemic. In like fashion, cholera, enteric fever, and dysentery in all of which the drinking water is usually at fault, can equally be conveyed by milk, vegetables, direct infection, etc.

5. The drinking water theory is further in conformity with the views of those who, while they hold that the "causa causans" of endemic goiter is as yet undiscovered, still admit that contaminated drinking water aggravates goiter.

6. Apart from water supply, inbreeding is a predisposing factor in cretinism and deaf-mutism.

7. The theory of the causation of goiter through deficiency of iodine cannot be accepted.

(a) Because even where iodine is present in excess (seacoast and sea) goiter may develop.

(b) Because deficiency of iodine causes atrophy, not hypertrophy of the thyroid.

8. Iodine is merely in some sort an antidote to goiter, as is quinine to malaria. Deficiency of quinine does not give rise to malaria, neither does deficiency of iodine give rise to endemic goiter.

9. Drinking water theory has much to recommend it from the side of prophylaxis as leading to an improvement of the water supply.

ENDERLEN: In college, I usually name the different theories (water, iodine insufficiency, infection) but in the end I must always confess that we know nothing definitely. Probably there are different causes at work. The result of the goiter commission in Berne in the year 1927 leaves us more or less in ignorance.

KLOSE: As I see it, we do not yet know the probable cause of goiter. Experiments have shown us that the growth of goiter is dependent on telluric and cosmic conditions. Important are the geological and water conditions, also the air. It is surely not insufficiency of iodine, as we here in Danzig have an over-rich goiter district, even though the air is richer in iodine than anywhere else on earth.

We must strongly differentiate the diffuse goiter from the knotty goiter. The diffuse goiter is a hypertrophy, the knotty goiter is a tumor. In the different countries the respective goiters will make their appearance quite differently. For instance, we have here in Danzig the small knotty goiter very profusely, where as in the mountain regions, the large knotty goiter is more in evidence. It also seems that in Germany, goiter is more prevalent in certain river valleys, for instance, the Rhein and Main districts bring forth a very definite goiter, so that undoubtedly water and air are helpful in the creation of goiter. Researches of air, water and earth in those districts are so far not at hand; I can only say that here, along the shore of the East sea, the air is very richly impregnated with iodine. For that reason, we cannot in our district, give iodine for the elimination and treatment of goiter.

The diffused goiter is the foundation of the Basedowic sickness; the knotty goiter can also get Basedow infected, yet, however, less frequent; for that reason there must always be made a distinction in the treatment of the Basedowic sickness, as to whether it concerns a diffused goiter or a secondary Basedow infected.

Also there are lacking sufficient researches into the existence of goiter in families. The American Goiter Congress would earn itself great praise, if it would, along with the numerous goiter problems, give new impetus especially for experimental research.

HIS: I am not in position to give you an answer on your question. The International Goiter Conference at Berne has surely shown us that heretofore we have had no satisfactory explanation of goiter etiology. Certain it is,

that the iodine content of water and food is not the only cause. Investigations I have had conducted in Prussia have shown for goiter-free and goiter districts about the same iodine figures.

BRUNNER: I am a firm believer of the theory of the insufficiency of iodine, as that, of all known theories, according to my conception, will explain best the differing appearance of goiter. Of course, I also admit the possibility that besides, other factors, for instance hygienic causes in general and pollution of the drinking water specifically, can play a certain rôle. However, of that we possess so far only scattered positive knowledge. On the other hand, in districts where iodine-containing salt was introduced in large measure, the showing in reduction of goiter cases, especially in the new-born and children, was so surprisingly large, that we cannot skeptically disregard the great influence of iodine. I proclaim firmly that personally I have seen no case of iodine injury, where iodine containing salt has raised a question of being the primary cause of the hyperthyreose. I am of firmest opinion that in goiter districts, similar to Switzerland, the sickness can only be successfully opposed if iodine-containing salt shall be lawfully introduced. Such a proposition can be made, as careful researches have shown that iodine injuries through salt, which were for a time often talked about, are practically non-existent.

KOCHER: The question of the cause of goiter is no more solved today than it was fifty years ago. At any rate all the etiologic factors that have been established at that time are still acknowledged as such, and the discussion of the different theories is still going on.

It seems very important for the question that nowadays studies and researches have been made in different parts of the whole world, and not only in countries where goiter is endemic. From all these studies, researches and investigations we can draw the conclusion that there is more than one unique factor existing in the etiology of goiter.

For the exogen origin, Theodore Kocher was the first to incriminate the water in countries with goiter endemicity and up to date this is the general opinion, but as to the goiter-causing agent in the water, the opinion differs a good deal, one from the other. While some believe in an infectious goiter germ in the water, others believe in a chemical agent, some combine these two theories. There is no doubt

that the theory which incriminates the deficiency of iodine in the salt and water, is the best founded, and a few years ago, I wrote in the textbook of Kaus and Drujsch that the cause of goiter is a disturbance of the metabolism of the thyroid gland more especially of the iodine and albumen metabolism. However, the deficiency of iodine and its influence on the metabolism of the thyroid cannot be the only cause of goiter formation, as it does not exist everywhere where goiter occurs. The problem is not as simple as that. The importance and significance of iodine in the physiology of the thyroid gland and in the causation of goiter represents only one part of the problem. It is the part of the physiology of the thyroid which alone is well known to us at present. It is certain that besides the iodine, several other chemical substances, which are in close connection with the metabolism of the thyroid gland, also play an important part in the causation of goiter; we only mention calcium and phosphorus.

Finally heredity plays a very important part in the causation of goiter. The fact that the goiter-causing agents act already on the thyroid of the fetus in utero does not exclude the hereditary transmission of an etiologic disposition to the formation of goiter.

CRILE: Function of thyroid gland is fabrication of iodine into an organic compound which exercises a basic control over the bodily processes. Endemic goiter is a geologic deficiency disease due to a lack of iodine in the organism.

OLESEN: I am glad to give you my ideas, but must warn you that I follow so closely the teachings of Marine that he, and not I, should be credited for the conceptions.

Endemic or simple goiter is due either to an absolute or a relative deficiency of iodine.

1. Absolute deficiency: An absolute deficiency implies an absence of iodine from food and water customarily consumed, or a decrease in the amount of available iodine so that an insufficient quantity is available for maintaining the equilibrium of the thyroid.

2. Relative deficiency: a. During certain periods, such as puberty, pregnancy, lactation and the menopause, there is an increased demand for iodine. Unless the element is available in more than normal quantities, thyroid hyperplasia may result.

b. When there is interference with the intake and utilization of iodine available in normal

quantities, thyroid hyperplasia may result. This may occur in infections, intoxications and during the use of abnormal food combinations (excess of fat or excess of white flour).

DUNHILL: 1. Deficiency of iodine, not necessarily in the intake of iodine, but in the amount ultimately available for the thyroid gland. In some individuals what is ingested may, for many reasons, fail to become available.

2. Some food defect, not necessarily vitamins.

3. Infections.

None of these factors have been shown to cause hyperthyroidism or toxic symptoms, but they may well be predisposing causes, and if when these are present in an individual there occurs:

4. Psychic trauma.

5. Sex imbalance.

6. The x-factor which determines why in any individual the machinery gives way, while in a number of his fellows similarly situated, it does not.

KINARD: As to the etiology of goiter, I believe there are different causes for different forms of goiter. I believe in this country all forms of goiter are merely different phases of a continuous pathological process as Marine has so sensibly worked out after the suggestions of Von Wegelin. I think heredity, environment, stress or strain (nervous and sexual), infection, and perhaps the underlying factor of real importance—lack of iodine.

MURRAY: Considers sex, family predisposition, sudden and prolonged strain, deficiency of iodine, the more important factors in the etiology of goiter.

SIR JAMES BERRY: Conclusions: 1. Simple endemic goiter is not a hypertrophy but essentially a degeneration of the thyroid gland. The gland is not overactive but underactive.

2. Whatever may be the connection between iodine and the thyroid gland, there is no reason for believing that a lack of iodine has anything to do with the causation of endemic goiter, as found in the human subject.

3. It is quite certain that at least in the majority of the cases, the disease is produced through the agency of drinking water.

4. There is much evidence that practically all waters which produce goiter contain frequently, although not necessarily at all times and seasons, mineral matter in suspension, usually in extremely fine state of subdivision.

5. There is also a good deal of evidence that this mineral matter is generally of a calcareous nature.

6. Organic matter in suspension, although capable apparently of causing a hyperplasia of the gland, at least in animals, has not been proved to be the cause of endemic goiter as seen in man.

GRAHAM: 1. Primary factor. An insufficient amount of iodine in the air, water and food to maintain the thyroid in a state of normal iodine saturation.

2. Secondary factors (a) Anything that interferes with the capacity to utilize iodine when present in sufficient quantities. (b) Anything that increases the physiological requirement in individuals whose natural iodine intake is sufficient for ordinary or normal needs but not sufficient for both the normal and the excess demand. Very concisely, the foregoing is what I have accepted tentatively as to the etiology of goiter. Naturally it applies only to endemic goiter.

JACKSON: The results which I have obtained with iodine therapy have somewhat altered my viewpoint. It now appears to me that there must be some other factor besides a mere deficiency of iodine that is responsible for the development of the simple or so-called "colloid" type of goiter. I believe that a microorganism is possibly the other factor. I feel that adenomatous goiter develops as a form of compensatory hypertrophy in neglected colloid goiters. Recently I have completed a study of the etiology of 300 cases of exophthalmic goiter. In many of these, infection seemed to be the factor. In others, there was the usual history of undue mental or physical strain.

KING: My opinion in brief is that adenomatous goiter is congenital in its nature and subsequently stimulated to growth by iodine deficiency; by infections; pregnancies; mental and physical stresses; et cetera.

The exophthalmic goiter does not depend on inherited tendencies nor iodine deficiency, but depends upon nervous, mental, and emotional disturbances; physical trauma and shock; infection, pregnancies, and various physical and mental stresses.

The colloid goiter is a pure iodine deficiency disease which has as additional factors, growths, infections, pregnancies, etc.

SLOAN: Hyperplasia occurs following inflammatory reaction due to some infection. I believe that adenomatous goiter is the end-

result of simple colloid goiter. In my opinion the cause of simple colloid goiter is mineral deficiency in the body; the cause of exophthalmic goiter in some infection.

BRAM: It appears to me that all these observers are right and that endemicity of goiter depends upon one agency here, another agency there, and still a third agency elsewhere. In other words, endemic goiter is a symptom, and as other symptoms, may be produced by more than one cause. It appears probable that in this country the major cause is iodine deficiency in air, food and water; in England, calcareous deposits in the soil and water; in India, unhygienic conditions; and everywhere one or more of these three in varying degree. In endemic as well as any other form of thyroid disease, heredity plays its part.

Sporadic thyroid enlargement is also due to many causes, the best known of which are heredity, physiological changes incident to growth of body and mind, focal infections, undue physical and mental stress and strain, under- and malnutrition, and the acute infections.

The causes of thyroid adenoma and cystic goiter (rarely occurring before the age of twenty-one) are usually traceable to neglect and misinformation regarding the treatment of the hypertrophic or colloid goiter which preceded. The cause of so-called toxic adenoma or hyperthyroidism is generally unknown.

The cause of exophthalmic goiter or Graves' disease is still an enigma to most clinicians. Bram believes that the disease is not goiter at all but a constitutional affection in which the thyroid gland, when enlarged at all, is a defensive reaction endeavoring, to protect the body against known and unknown toxins originating elsewhere in the economy. Bram believes that exophthalmic goiter is a misnomer in which neither thyroid enlargement nor exophthalmous may be present in an otherwise typical syndrome, and prefers to use the term Graves' disease, since it is noncommittal in regard to eyes and thyroid gland. The fact of the matter is that the cause of exophthalmic goiter or Graves' disease is yet to be discovered although the neuroendocrine theory based on individual susceptibility as predisposing, and psychic trauma as the exciting cause, appears plausible.

TIFFIN: It is my firm conviction and belief that iodine deficiency is at the base of the disturbance in the more common type of

goiter. I am firmly convinced that in the hyperplasias of thyroid disease there is an associated infection but that this is dependent on the iodine deficiency; particularly do I feel that this is true in the latter stages of exophthalmic goiter, as is clearly shown by the clinical findings the so-called crisis is typically an infection process, and we know, the causes of death are practically always in these cases found to be caused by pneumonia or some other "itis."

DE JOSSELIN DE JONG: When speaking of goiter as a prevalent disease of mankind in different parts of the world, I wish to exclude all those enlargements of the thyroid gland that are due to: cancer and other malign blastomata, syphilis, tuberculosis, inflammations, hematoma.

As for the etiology of the endemic goiter it is my opinion that this problem cannot be solved by admitting one single cause. The problem seems to me to be a very complex one and in this I wholly agree with the words of Wegelin (Berne): "The etiology of the endemic goiter is very complex and contains exogen as well as endogen influences."

As important endogen factors I regard: sex, age and constitution (viz. the rôle of the whole endocrine system, heredity). As for the exogen factors, I consider the lack of iodine (in eatables, drinkables, and the air) as an important factor, but surely not the only one, which can explain all.

The examination of the drinking water: Little iodine was combined with much goiter; much iodine was combined with few goiter cases.

Goiter is a generic term which includes a variety of diseases of diverse etiology. Future research may, and no doubt will, further subdivide the simple goiters clinically, pathologically and etiologically. But meanwhile we have the means of preventing large numbers of them by attention to the fundamental principles of nutrition and of personal and social hygiene. Gastrointestinal infection may not in itself cause chronic hypertrophic goiter in the presence of a sufficiency of iodine, nor may an insufficiency of iodine in itself cause chronic hypertrophic goiter in the absence of gastrointestinal infection; but when both are present, the conditions for the development of this type of goiter are at their optimum.

I do not consider infection, however, to be a cause of goiter. Experimental investigation

has been made. Now I can tell you that, experimenting with feces and with cultures made out of feces of goiter patients, we have been unable to produce an enlargement of the thyroid gland of any importance in rats. These experiments have been continued throughout several months, but without any results. So I cannot agree with those who consider infection as the most important cause of goiter. Bacteriological examination of several specimens of goiter of men has not given a positive result contra-Folley.

So briefly resuming, I can give as my opinion.

1. The etiology of endemic goiter is a very complex problem. One should be careful not to consider the problem too much from one point of view.

2. Endogen and exogen influences of different kinds may be of smaller or greater importance for the origin of goiter. As endogen factors I must consider: age, sex, constitution. As for the exogen influences see what follows.

3. The influence of the lack of iodine in eatables, drinkables, and the surrounding air cannot be denied.

4. That goiter is caused by infection (digestive tract, bacterium toxins, etc.) has not been proved until now. My experience and the result of experiments, made in my institute, have not given me the conviction that infection is an important factor among the causes of goiter.

5. What we want is a well-founded knowledge of the function of the normal thyroid gland, as well as of the endemic goiterous gland.

6. And finally: Goiter and goiter are not always the same; it is possible that the so-called endemic goiter in different parts of the world is caused by a complex of different influences. Is it possible to consider the goiter in Switzerland and in Holland, in the Himalayas and in the northern part of Italy, in Sumatra and in Siberia, etc. etc. as the result of the same complex of etiological factors? Here too, I feel a lack of knowledge.

ELSE: (1) Iodine deficiency is the primary cause of most goiters.

(2) Iodine deficiency may be absolute or relative.

It is absolute when the iodine intake is not sufficient to produce the required thyroxin. It is relative when the iodine intake is sufficient but the iodine digestion is deficient.

(3) There is a small group of goiters that

result from deficient thyroid glands even in the presence of sufficient iodine.

These may be the result of a congenital defect or an acquired defect in later life.

CROTTI: Cause of endemic goiter is not lack of iodine, as is so much spoken about, but a direct infection which is transmitted to the thyroid gland through the water supply. This infection is isolated by the gastrointestinal tract and then settles in the thyroid gland.

So far as toxic goiter is concerned, I think we have to deal with a toxic thyroiditis which is caused by many infections such as grippe, tonsillitis, and a great many others. When once the thyroid gland is affected, the secretion and function of the gland becomes disturbed and the ailment resulting is a thyrotoxic condition.

BERRY: Endemic goiter is not caused by lack of iodine in soil, water and food, but it is produced by some deleterious agent in the drinking water. There is much to be said for this agent being of an organic nature, but the proof of this is to my mind still not quite complete; and I have but very little doubt that inorganic impurities also play an important part in the causation of the disease.

DE QUERVAIN: Intoxication of different kinds, insufficiency of iodine and perhaps of vitamins in the food and heredity may be the three most important factors.

PLUMMER: The etiology of goiter is a subject regarding which I have never attempted to express my opinion in print further than the point of accepting iodine as a factor in the cause of endemic goiter. There are many contributing factors like sex, age, etc. to be considered. I am of the opinion that there are other unknown factors. I have discussed the possibilities and theories of exophthalmic goiter in private conversation, always ending up the discussion with the statement that I do not know anything about the cause of exophthalmic goiter. Furthermore, I am positive in my opinion that what we have designated as hyperfunctioning adenomatous goiter has a different etiology from exophthalmic goiter. I am quite confident that they have certain contributing factors in common.

MARINE: To summarize, however, my views on the etiology of simple goiter very briefly, I believe that the immediate cause of thyroid enlargement is due to a relative and occasionally an absolute deficiency of iodine, while the remote, underlying or fundamental cause is

still unknown. This unknown factor I have been in the habit of speaking of as the factor x. I believe it is a positive definite substance rather than a lack of something which creates increased demands for iodine. The idea that this factor could be a specific infection, that is a contagium vivum, I believe to be untenable. That the substance might arise as a result of bacterial growth one must admit, but it must also be admitted that a great many other plants and possibly animals can produce it. This factor x in all probability is a definite chemical substance and it probably acts indirectly on the thyroid by increasing the needs of the various body tissues for thyroxine.

Regarding the etiology of Graves' disease, which I believe to be a much more complex and in many respects a quite different condition from simple goiter, I have nothing new to add beyond my long expressed opinion that the thyroid plays an entirely secondary rôle in the disease. Fundamentally the disease depends upon an inherited or acquired constitutional defect depending upon a hormonal imbalance of the visceral nervous system, and in which a deficiency of some function of the suprarenal cortex and gonads play an important rôle.

FAHRNI: I might summarize by saying that I am favorable to Marine's views and that I think it most probable that colloid goiter in general is the result of the reaction on the part of the thyroid to an insufficient absorption of iodine. Unquestionably, McCarrison's views are contributory causes in many cases at least.

In regard to the adenomata, I might say in a general way that in my opinion these growths are largely, probably entirely, secondary to earlier colloid goiter manifestations in the thyroid gland, and I feel that the prevention of colloid goiter will to a very large extent, at least, prevent the development of adenoma.

Graves' disease is different, of course, and while I feel that this disease usually develops following some shock to the central nervous system or in an individual whose resistance has been greatly lowered as in influenza infection, at the same time there is no question but what iodine metabolism plays a big part also.

HUTTON: I have never believed that the thyroid was the only thing at fault in Graves' disease, or even that it was the principal offender. I am convinced that the adrenals are intimately involved in this question, and I think, ere long, one or more hormones will be isolated from its cortex. As a matter of fact,

some work is already being done along that line, but it is not yet ready for publication.

HERTZLER: "As is true of other chronic diseases, we do not know the cause of goiter. We can but detail some of the conditions and circumstances under which it arises. The knowledge we possess relative to the causation is of general interest only." Following this statement, he discusses all suggestive causes of goiter: heredity, iodine deficiency, infection, etc.

McCARRISON: The constellation of causes which give rise to goiter is not always the same, nor is the character of the disease identical in different parts of the world.

In endemic goiter McCarrison recognizes two theories: deficiency of iodine and infection, and personally believes that the truth lies in a judicious blend of both.

Lymphadenoid type: To the physician or surgeon who has to deal with goiter in the human subject, the points of interest arising from these observations are: (1) Lymphadenoid goiter occurs in man; it can be produced under experimental conditions in rats. (2) It arises in approximately 25 per cent of young rats despite the adequate ingestion of iodine. (3) Deficiency of iodine in the food is certainly not the cause of lymphadenoid goiter as it occurs in rats; it is unlikely, therefore, to be the cause of lymphadenoid goiter as it occurs in man, and the additional provision of iodine will not prevent it. (4) The basal factor in the causation of lymphadenoid goiter in rats is a dietetic one; it may reasonably be expected that the basal factor in its causation in man is likewise dietetic and of a like order. (5) Judging from experimental experience in rats the diets which are likely to be associated with lymphadenoid goiter in man are those composed largely of white flour or other vitamin-poor fats and in which there is a paucity of fresh fruit and green leafy vegetables. In view of the almost universal use of white flour as the main staple of the dietary in western countries, of the widespread use of vitamin-poor carbohydrate foods, of substitutes for butter which are relatively poor in vitamin A, of the use also of frozen and tinned meats, and of the small proportion of fresh fruit and green leafy vegetables in many people's diets, the etiological significance of the observations made on rats appears to me to be great.

LEON ASHER: As a physiologist, however great my interest in goiter is, I have no experience in the pathology of the thyroid gland, only on the physiological aspects of this ques-

tion. With this restriction, it appears to me that there is very much evidence in favor of the view that lack of iodine belongs to the fundamental causes of goiter. I should not like quite to exclude the possibility that there are cases of goiter where the deficiency of the thyroid gland is only one very striking symptom of a more general and constitutional disease affecting many functions and tissues of the human body. For such an extended constitutional disease there might be a deeper cause than mere lack of iodine.

LOBENHOFFER: I would summarize my views as follows:

1. Endemic goiter approaches the infectious diseases.

2. An external cause is to be sought in a probable organic poison, which though being local, is so not to such an extent as to make impossible fluctuations in extension and intensity; the telluric etiology can, therefore, not be maintained.

3. Cooperation of several external and internal moments is necessary to allow endemic goiter to arise.

4. Iodine deficiency alone cannot be considered as a cause of goiter.

5. In order to further the researches on goiter it seems necessary to make in a greater measure than hitherto very minute individual investigations in the endemic districts.

BREITNER: As for the question of etiology, the diagnosis is the end of the dispute about uniform etiology. Different causes can be effective at the same time. The dispute about the water theory, the iodine theory, the dirt theory, the infection theory, and all other theories loses its monopoly. No one alone, but every one is right in some respects.

VAN METER: I have for some time believed that the etiological factor or factors in the common types of goiter were in some manner dissimilar, varied in intensity or were altered by some unrecognized influence in different parts of this country.

ADLERCRENTZ: Believes that endemic goiter is probably due to many factors which may vary in different localities.

KIMBALL: Believes that endemic goiter is a deficiency disease. He also thinks that the etiology of hyperthyroidism lies within the individual and feels that these etiologic factors are increased or aggravated by endemic goiter.

WILLIAMSON AND PEARCE: In toxic hyperplasia the essential etiology is an affection of

the lymphogenic activity of the thyroid. Non-toxic hypertrophy, infection, contamination, infection not acting on the thyroid but on the intestinal threshold (gut). Mere iodine deficiency will not explain endemic goiter. Lack of the mobilizer of iodine-lymphogenic secretion, is an hypothesis which satisfies the facts.

MUGGIA: I believe that goiter depends on alimentation; an indisputable parallelism exists I believe, from the epidemiological point of view, between pellagra and goiter. In more exact terms, I believe that goiter is a disease of insolvency. But the efforts that have been made of demonstrating that it depends primarily on deficiency of iodine, do not seem to me to have met with true success. As for the lack of iodine in the organism of those suffering from goiter, it is not due, in my opinion, to a lack of iodine in the air, in the water, or in the food; but to the incapacity of the thyroid, previously injured by other causes (avitaminosis) to assimilate it. On the contrary, the improvement observed during recent years independently of all curative intervention, proceeding simultaneously with improvement in alimentation, leads us to conclude that goiter is perhaps with us merely an avitaminosis. I say "with us" because it is possible that the causes of goiter are not everywhere the same. Moreover, the parallelism observed by v. Fellenberg between the quantity of iodine and vitamins of certain foods seems to come to the support of our view which is clearly defined by Maranon; a fact which is not without importance since Spain as well as Italy has well recognized the calamity of pellagra. Better than elsewhere, I believe that one can understand the epidemics of goiter in barracks and boarding schools.

BLUM: I regret to have to give you as much negative as positive information. I oppose the theory that iodine-deficiency is the cause of goiter. Not only have the doubts that I expressed regarding the Colorimetry of smallest iodine quantities been justified and acknowledged but I have recently ascertained that the first part of the analysis according to von Fellenberg, fails utterly. From the histological point of view, it is not as easy to pronounce this theory as incorrect, as it is from the standpoint of physiological chemical research. The histological reports on "Proliferation of the Hunger-thyroid-Gland" go to prove that a disturbed internal metabolism, such as is caused by the hunger condition (biliary pig-

ment in the urine, albuminuria, etc.) can influence the thyroid gland and cause goiters. The normal blood possesses therefore a very considerable antithyroidal power. The blood of Graves' disease patients acts on the metamorphosis of the tadpoles in the same way as thyroidal substance. If this observation is correct, then in the first place the blood of Graves disease patients must have lost its peculiar antithyroidal power. Keeping this in mind, the question naturally crops up as to whether the antithyroidal power of the blood is not the moving principle in the disappearance of the swelling. I call to mind the type of goiter to be found in places where iodine abounds (i.e. the sea-coast) and which shows an endemic character. If one recollects that most of the endemic struma discovered, is in no way comparative to their size in general, contain positively more iodine than normal glands, one must admit that the iodine deficiency has nothing to do with the origin of goiter, but that obviously a "genius loci" was here at work, in the community where the patient lives. On the other hand, there can be no doubt that "goiter-water," effective as it is in experiments with animals, can be rendered harmless through boiling. This fact forces one to believe that an active virus, passing with food into the digestive organs, from there sends its harmful products into the system as goiter-noxe.

BERAND AND DUNNETT: Altitude and climate play only a subsidiary part in the genesis of goiter. Goiter has decreased in intensity (notwithstanding) even though no systematic preventive treatment with iodine has come to light, and assume the cause of the decrease to be due to improvement in drinking water supply, and increase in consumption of wine; decrease in number of marriages between blood relatives; improvement in general living conditions. Drinking water plays a most important part. There are beyond all doubt certain kinds of water which produce goiter. They consider goiter-producing water acts by interference with iodine metabolism; consequently all factors that increase the need of the system for iodine favor the appearance of goiter, and they mention puberty, pregnancy, lactation and the climacteric period. They do not consider goiter as an affection strictly confined to the thyroid. They confirm the opinion that carcinoma in the thyroid gland occurs as a result of already existing struma.

[NOTE: For bibliography see Author's reprints.]

INTERCHANGEABLE TYPES OF GOITER*

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WE have been led to believe that the different types of goiter represent definite clinical entities, and the that histological findings coincide with the clinical pictures. The classification of goiters which is most commonly used is: (1) adolescent; (2) colloid; (3) adenomatous, with and without hyperthyroidism; (4) exophthalmic goiter or Graves' disease. This classification was generally accepted until the indiscriminate use of iodine and thyroid medication made the above classification questionable, both clinically and histologically.

We owe to Marine and Williams⁴ our knowledge of the changes produced in exophthalmic goiters by iodine administration. In 1908 they reported 4 cases clinically regarded as exophthalmic goiter of the mild type which had, however, been treated with iodine, and were all anatomically pure colloid goiter, and they state there is a return to the colloid type on the administration of continued minute doses of iodine, even in exophthalmic hyperplasia. Plummer⁶ in 1922 introduced iodine in the form of Lugol's solution for preoperative and postoperative use in patients suffering from exophthalmic goiter. He differentiated between adenomatous goiter with hyperthyroidism and exophthalmic goiter, with the understanding that Lugol's solution was specific for the exophthalmic type but not in the adenomatous goiter. His views were generally accepted until Graham¹ in 1926 published the results of his work, which revealed that Lugol's solution was effectual in reducing the thyrotoxicosis and metabolic rate in both the adenomatous and exophthalmic types, and stated: "It is a matter of considerable importance to recognize that the quantity of iodine necessary to bring about the same

or comparable clinical response and decrease of basal metabolic rate is much less in cases of toxic adenoma than in cases of exophthalmic goiter. This we attribute to the difference in degree of hypertrophy and hyperplasia of the thyroid in the two conditions." His work was followed by Rienhoff's⁷ which gave us a clearer conception why iodine was beneficial in the two types. He stated that "From the evidence at present, it would seem that most of these tumors palpable in nodular goiters are nothing more than involutinal bodies of the same type as have been described, and that a great many, in fact nearly all of the cases of nodular goiter with or without hyperthyroidism which have been described as colloid adenomas, mixed fetal and colloid adenomas, colloid cysts, cystic adenomas and miliary adenomas are in no sense of the word adenomatous but the result of an attempt on the part of the thyroid gland, following an hypertrophy, to reapproximate its normal histologic structure, namely, involutinal bodies or areas of hyperinvolution and hypo-involution."

Mosser⁵ offers a very satisfactory explanation for the temporary improvement following iodine administration in cases of hyperthyroidism. He states: "When iodine is first given the cells are stimulated to secrete an excessive amount of colloid. This colloid fills the acini and mechanically compresses the lining cells, thus reducing their secretory power. Less thyroxin is produced, and the patient shows clinical improvement. Gradually the cells adjust themselves to the changed condition and resume their secretory power. The amount of thyroxin is thus again increased and the toxic symptoms increase proportionately. Further iodine medication fails to alter the

* Read at the Annual Meeting of the American Association for the Study of Goiter. Dayton, March 25-27, 1929. I wish to thank Dr. Nicholas M. Alter, Assistant Professor of Pathology at the New York Post-Graduate Medical School and Hospital for reviewing the sections.

production of thyroxin, but does continue to stimulate colloid production. After prolonged iodine administration the cells become exhausted, can no longer produce colloid, and on continual iodine stimulation they degenerate. However, even in the stage of exhaustion, they are still quite capable of carrying out their pathological function, i.e., production of excessive amounts of thyroxin. The microscopic picture, which is usually interpreted as a specific effect of iodine on the thyrotoxic producing properties of the cells, is in reality the effect of prolonged and excessive colloid production."

From the accumulating evidence we are being forced to change our conception of the different types of goiter and Hertzler³ helps to clarify the confusion, stating: "It was only after an accumulation of hundreds of specimens which presented unmistakable microscopic evidence that the goiters were of long standing that it occurred to me that something could be gained by securing a careful history covering the period antedating the time of the alleged origin of the goiter. Since going further back than the suggested date in the history, it is uncommon to find patients with an alleged sudden onset of the condition who do not give evidence of disturbances antedating the time given."

And Hellwig¹ states: "I am forced to the belief that exophthalmic goiter develops usually in a diffuse colloid goiter. . . . Likewise the distinction usually made clinically between innocent colloid and exophthalmic goiter does not seem to be justified, since the diffuse colloid goiter, at least the proliferating form is often associated with slight symptoms of hyperthyroidism."

The following cases do not coincide clinically and histologically and are considered atypical or interchangeable, if we are to believe that the different types of goiter represent separate clinical entities.

ADOLESCENT GOITER

CASE 1. Female, sixteen years of age, was examined in June, 1928 on being discharged

from an orphanage and was found to have an adolescent goiter. She did not know this condition existed until informed by the examining physician who advised her to take 2 drops of tincture of iodine in water daily. This she did for two months after which time she became quite nervous and had tremor of her hands. The iodine was discontinued but the patient did not show any improvement and was first seen by me on November 20, 1928. Examination revealed a definite enlargement over the thyroid, with a thrill and marked tremor of hands, and the patient was extremely nervous and restless. Basal metabolism November 24 was normal. She was advised to remain out of school and put on a high caloric diet and forced fluids, with elixir luminal drams 1, t. i. d. She was next seen on January 22, 1929. During this time she had lost 6 pounds in weight and her condition instead of improving had become worse. Hospitalization was advised and her basal metabolism on January 24, 1929 was a +3. After being put on Lugol's solution, minims 20, luminal grains $1\frac{1}{2}$, ovarian substance grains 5, and pancreatic substance grains 2, t. i. d., the patient showed marked improvement. Thyroidectomy was done January 30, and the patient made an uneventful recovery. Pathological diagnosis: Exophthalmic goiter. (Fig. 1.)

COLLOID GOITER

CASE II. Female, thirty-three years of age, stated that in September, 1927 she consulted her family physician for a swelling of her left ankle and a goiter. At that time she was found to have a phlebitis of the left ankle. On communicating with Dr. Felder, her family physician, I found the patient had a colloid enlargement of her thyroid, without symptoms. The patient was not given any thyroid or iodine medication but five months later, as her mother had died from an exophthalmic goiter, she consulted a thyroid specialist in New York, for she was rather conscious of the slight fullness in her neck. There were no symptoms referable to the thyroid at that time. Basal metabolism February 4, 1928 was -1. She was informed that she had no thyroid disturbance but was given Lugol's solution minims 3, t. i. d. Three months later she had lost 8 to 10 pounds in weight, her eyes were enlarged and she was nervous and quite irritable, and bothered with palpitation. She

consulted another physician who told her she had Graves' disease. The patient was given iodide of mercury grains 1, o. d., and advised

age, a nurse, was first seen by me on August 4, 1927, stating that twelve years previously she had been operated on for an adenoma of



FIG. 1.

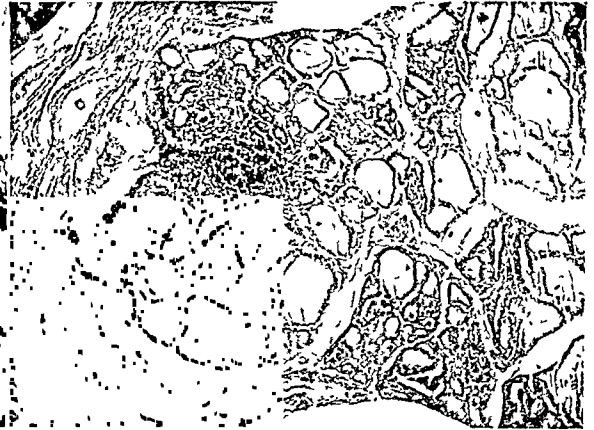


FIG. 2.

to have roentgen-ray therapy. She received several treatments and showed improvement but four months later, not feeling entirely well, she consulted Dr. Carter, at which time her weight was 118 pounds, as against her best weight of 135 pounds. Basal metabolism on October 27, 1928 was 3 below the average normal. I saw this patient in consultation

the isthmus of the thyroid. About one year after this operation, she noticed a lump in the right side of her neck. This had increased slightly in size and about seven weeks before consulting me she had a gastrointestinal upset from eating sea food. Since that time she had lost 20 pounds in weight, and had palpitation of her heart and shortness of breath on

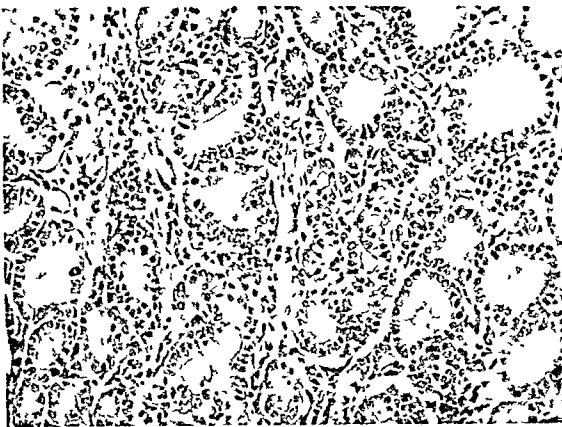


FIG. 3.



FIG. 4.

with Dr. Carter on October 28, 1928 at which time there was definite evidence of enlargement of the thyroid with a thrill over it, and it was quite apparent that the patient was suffering from an exophthalmic goiter. Thyroidectomy was performed on November 5, 1928, by Dr. Carter. Pathological diagnosis: exophthalmic goiter in resting stage. (Fig. 2.)

ADENOMATOUS GOITER

CASE III. Female, thirty-eight years of

going up stairs. Otherwise she felt well. Her menstrual periods had been scanty during the last few months. Examination: There was no evidence of exophthalmos in the right eye. The left eye had been enucleated following an accident at the age of two years. At the time of examination there was a nodular mass involving the right lobe without a thrill and her pulse was 108. A diagnosis of adenoma of the thyroid with hyperthyroidism was made. Basal metabolism on August 5 was +45. The

patient was advised to enter the hospital for operation, which she did, and was operated upon on August 22, 1927. She made an unevent-

made and basal metabolism on December 2, 1927 was +3. Patient was informed she had the type of goiter that could not be treated

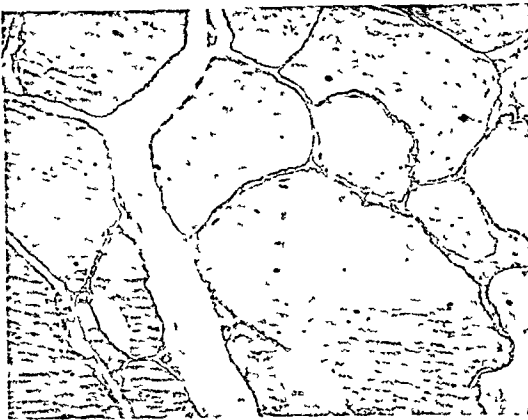


FIG. 5.



FIG. 6.

ful recovery, being discharged from the hospital on August 30, 1927. Pathological report: hyperplastic goiter of Graves' type, in a stage of slight remission at the time. (Fig. 3.)

CASE IV. Female, twenty-six years of age, was first seen by me on November 30, 1927 complaining of a swelling in her neck which

by medication, but as I had treated her sister for a colloid goiter with a satisfactory result, she demanded medication before submitting to an operation; hence she was put on thyroid extract, grains 1, t. i. d. The patient was next seen on January 18, 1928 at which time her weight was $137\frac{1}{4}$ pounds and pulse 90. She had no complaints but her neck remained

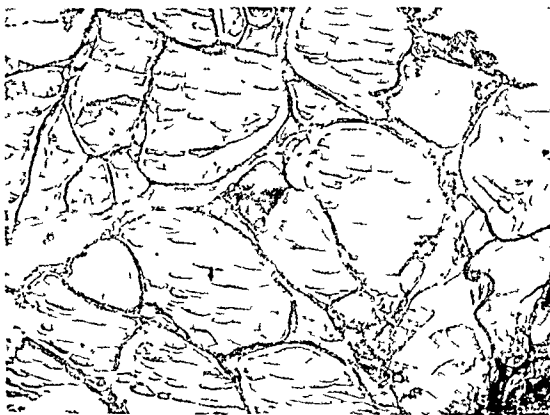


FIG. 7.

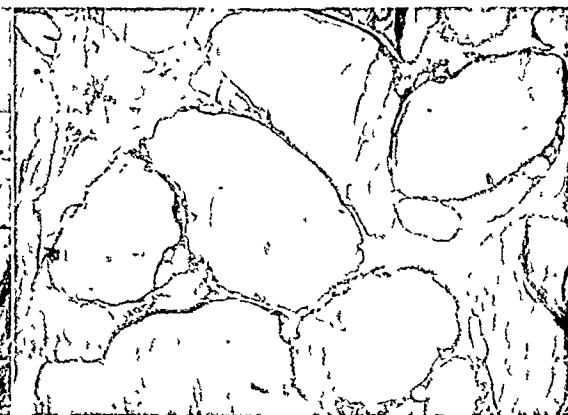


FIG. 8.

she had had for one year. Friends first noticed a lump in the right side of her neck, but at that time she had no symptoms referable to her goiter. Occasionally a sensation of pressure and choking were noticed, but otherwise she felt perfectly well. Examination was negative, with the exception of a definite mass involving the right lobe of the thyroid. The left lobe was negative. Weight was $133\frac{3}{4}$ pounds, pulse 100. Diagnosis of adenoma of the thyroid was

unchanged and she was given thyroid extract, grains 12, t. i. d. On March 21, 1928 she returned stating she had had nausea and vomiting and had been bothered with diarrhea for two weeks and was beginning to feel ill. She complained of nervousness and palpitation and was losing weight. Examination revealed an enlargement over the thyroid region with a definite thrill over it, and a beginning exophthalmos. Patient was advised to enter hospital

for observation and operation, which she did on March 22, 1928. Basal metabolism on March 26, 1928 revealed a +60. Weight was



FIG. 9.

105 pounds and pulse 160. The patient was operated upon on April 2, 1928 and made an uneventful recovery. Pathological report: exophthalmic goiter in the stage of remission. (Fig. 4.)

CASE V. Female twenty-six years of age, first seen on August 30, 1926, stating that she

the right lobe. No thrill or exophthalmos was found, and the pulse was 90. Basal metabolism August 31, 1926 was +4. Diagnosis: diffuse adenomatous goiter. Thyroidectomy was done September 4, 1926. Pathological report: Exophthalmic goiter in stage of remission. (Figs. 5 and 6.)

EXOPHTHALMIC GOITER

CASE VI. Female, aged twenty-six, first seen on January 17, 1927. She had been very nervous and irritable for the past year. In February, 1927 she began to have trouble swallowing, and at that time consulted a physician who gave her iodine to take, but after six weeks of non-improvement she consulted another physician who gave her nine roentgen-ray treatments. After this still another physician gave her violet-ray treatment and did a tonsillectomy, and also gave her medicine for her nervousness. She continued on this treatment until November, 1927 when being still unimproved the patient discontinued all treatment. Examination revealed a definite tremor of hands with slight exophthalmos, slight enlargement over the thyroid with a thrill, weight 118½ pounds as against a best weight 148 pounds, and pulse



FIG. 10.

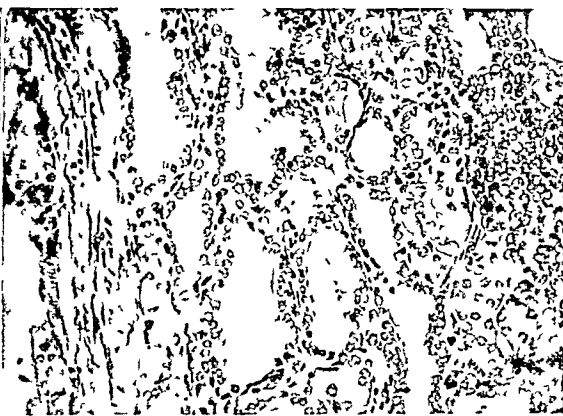


FIG. 11.

had been bothered for two years by swelling of her neck. Her neck had always been full but two years ago she noticed it was getting larger and with the increase in size came added difficulty in swallowing. There was no sweating, tremor of hands, diarrhea or palpitation. Examination revealed a well-developed and nourished woman not appearing ill. The neck revealed an enlargement of both lobes of the thyroid, the right lobe being larger than the left. There was a definite encapsulated mass in

140. Basal metabolism on January 25, 1928 revealed a +14. Diagnosis: Exophthalmic goiter. The patient was advised to enter the hospital for observation and operation, which she did on January 24, 1928 and was operated upon on January 30. Pathological report: Colloid goiter of unusually homogeneous structure. (Fig. 7.)

CASE VII. Female, nineteen years of age, first seen April 16, 1927, complaining of palpi-

tation of her heart, swelling of neck, enlargement of her eyes, extreme nervousness and loss of weight for a period of four months. She had been taking medicine including iodine which seemed to make her feel worse. Examination revealed a slight exophthalmos with a definite fullness over the thyroid and a thrill, with tremor of fingers and moist skin. Best weight was 130 pounds, present weight 117 $\frac{3}{4}$ pounds, and pulse 140. Diagnosis: Exophthalmic goiter. I advised the patient to enter the hospital for treatment and operation, which she did on April 26, 1927. Basal metabolism April 28, -4. The patient was operated upon on May 4, 1927. Pathological report: Colloid goiter. (Fig. 8.)

CASE VIII. Female, forty-four years of age, first seen June 2, 1927, complaining of nervousness, tremor of fingers, swelling of neck and protruding eyes, from which she had suffered for a period of five years. Her symptoms came on following the death of her daughter from pneumonia. The patient had had a cholecystectomy and appendectomy ten years previous to the time she consulted me; otherwise her history was negative. Examination revealed bilateral exophthalmos with symmetrical swelling of thyroid and thrill over it. The heart was fibrillating and the pulse could not be counted accurately. Basal metabolism on May 28, 1927 was +69. Weight was 157 pounds with pulse 150. The patient was operated upon on June 11, 1927. Pathological report: Exophthalmic goiter in a resting stage, with two small adenomata. (Figs. 9, 10, 11.)

It would seem that the classification of

goiters used in the past is of questionable value and in certain instances the type of goiter merely represents a stage in the cycle of the thyroid disease.

CONCLUSIONS

1. Each case of goiter should be treated as individual and not according to pigeon-hole diagnosis.
2. The classification used in the past for the different types of goiter must be considered interchangeable.
3. An accurate diagnosis cannot be made from the clinical and laboratory findings alone, but these must be studied together with histological findings before arriving at a final diagnosis.

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HYPOTHYROIDISM*

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SPORADIC cretinism was recognized by C. Hilton Fagge in 1871, though what was undoubtedly endemic cretinism was reported in 1848 by Dr. Hugh Norris. All of Norris' group possessed a goiter while the constant absence of goiter in Fagge's cases was striking. In the latter's collection was a case of a child eight years of age who developed hypothyroidism following an attack of measles. This led Fagge to conclude that such a condition could develop in an adult and need not necessarily be congenital. Although he had never seen such a case, he visualized changes in the soft tissues which stand up at the present time as characteristic signs of myxedema.

The first actual report of adult myxedema seems to be that of Sir William Gull in 1873. His description was essentially complete except that no mention was made of body chilliness or absence of sweating. He ventured no theory as to the cause.

Surgeons observed that a characteristic symptom complex developed following total extirpation of the thyroid gland and J. L. Reverdin, T. Kocher, and Felix Semon at about the same time (1882-83) recognized the cause of myxedema as due to thyroid deficiency.

Magnus-Levy in 1894 contributed vastly from a diagnostic standpoint when he demonstrated the existence of a low basal metabolic rate in myxedema.

Hypothyroidism for a long time was classified under three groups of cases: cretinism, myxedema, and operative myxedema or cachexia strumipriva. With the improved diagnostic procedures and the advent of thyroxin by Kendall in 1915,

much has been learned about the intermediate degrees of hypothyroidism and varying forms in which it may occur.

PRENATAL DIAGNOSIS OF CRETINISM

The theory has been advanced that colloid goiter is most likely to occur at a time when stress is laid on the thyroid gland, such as at puberty and during pregnancy and infections. With this in mind the basal metabolic rate has been studied during pregnancy with the hope of throwing some light on the etiology of congenital cretinism.

Sandiford and Wheeler¹ have reported most completely their basal metabolism studies on a woman observed before, during, and after pregnancy, covering a period of seventeen months. They concluded that their experiments coincide essentially with those of Magnus-Levy, Zuntz, Hasselbalch, Carpenter and Murlin, Root and Root, Rowe Alcott and Mortimer which all showed beyond question that the total energy production of a pregnant woman increases slightly, beginning at the middle of gestation and finally reaching a maximum of approximately 20 per cent above her basal value before pregnancy. It is their contention that there is no change in the basal metabolic rate of a woman during pregnancy and that this increase represents the heat production of the newly formed protoplasmic tissue composed largely of the fetus and to a less extent of maternal tissue. They had calculated the heat production of the fetus separately from the total heat production in the pregnant woman to come to their final conclusion.

In Figure 1 is charted the basal metab-

* From the Dept. of Internal Medicine, St. Louis Univ. School of Medicine. Read at the Annual Meeting of the American Association for the Study of Goiter, Dayton, March 25-27, 1929.

olic rates of a pregnant woman before and during pregnancy. Her basal metabolic rate on two occasions before pregnancy

desiccated thyroid substance daily. No goiter developed. As the chart indicates, there was no increase in the basal metab-

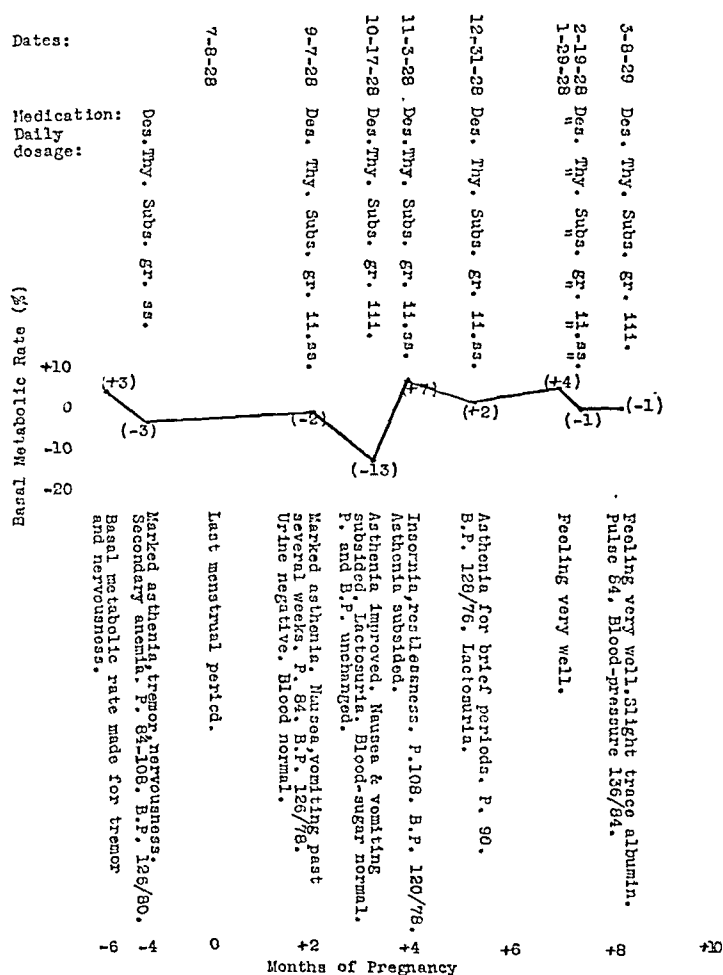


FIG. 1.

was within the range of -3 to +3 per cent. Because of marked asthenia complained of at the end of the second month of gestation, she was given thyroid medication and showed distinct improvement. She was found to tolerate $2\frac{1}{2}$ grains daily, though the basal metabolic rate was but -2 per cent at the time thyroid medication was instituted. This dosage was maintained and at the end of six weeks the basal metabolic rate was -13 per cent, or a decrease of 11 per cent. After the end of the second month of pregnancy, this patient received an average of $2\frac{1}{2}$ grains of

olic rate after the middle of gestation as Sandiford and Wheeler and the others had found in their experiments. If this patient had not been given thyroid substance, is it not reasonable to believe that her basal metabolic rate would have decreased materially? This case is also evidence that during pregnancy the thyroid function may either be reduced or fail to respond to the increased body surface and weight. Whether it is from such a type of case that the congenital cretin is born cannot with sound basis be said with the evidence so far at hand. There is

some clinical evidence to bear out such a suspicion in a case reported by Engelbach.² He found a low basal metabolic rate in a woman during the fourth month of gestation and administered desiccated thyroid substance to her throughout the remainder of her pregnancy. She gave birth to a child by cesarean section, weighing 7 pounds 8 ounces and normal in physical development as far as could be determined. This patient had previously given birth to two sons who were retarded mentally and physically, both of whom responded to thyroid and pituitary therapy.

This seems to be an advancement in determining the cause and thus preventing the occurrence of the dreaded and pathetic condition of sporadic cretinism. To establish its value would mean merely, (1) the determination of the basal metabolic rate of a sufficiently large group of women during pregnancy, and (2) the administration of thyroid substance to tolerance in those cases not showing an increased metabolic rate after the fifth to sixth month. If in so doing no case of congenital cretinism developed, the theory that congenital cretinism develops when the thyroid activity of the mother fails to respond during pregnancy would be established.

There will be opportunity to observe the child of the patient discussed in Figure 1 for any evidence of hypothyroidism. It will also be of interest to follow the mother through lactation by basal metabolism studies. This patient is one of those cases occasionally seen who have a normal basal metabolic rate but improve on thyroid therapy.

POSTNATAL DIAGNOSIS OF HYPOTHYROIDISM

In infancy, the thyroid hormone is of prime importance in the physical as well as the mental development. Since it is impossible to measure the mental development of an infant during the first months of life we must rely on observation of the physical development for an early diagnosis. By far the earliest means of detecting physical retardation in an infant is by

radiological examination of the osseous system as outlined by Engelbach and McMahon.³ They demonstrated the presence of four osseous nuclei in a normal child at birth. These are the lower epiphysis of the femur, the upper epiphysis of the tibia, and two tarsal centers, the talus and calcaneum. They have also outlined a table showing the time for appearance of the different nuclei in a normally developing child. It is their contention that a delay in ossification signifies uncomplicated hypothyroidism.

With advancing months, there are certain physical achievements which a normal child attains. These are listed in Table 1.

TABLE 1

Age	Developmental Stages in Normal Child	Suggestive Signs of Defectiveness
Birth.....	Weight between 7 and 8 pounds.	Weight over 8½ pounds.
10 days.....	Separation of umbilical cord.	Delay beyond 15th day.
4 mos.....	Able to hold head up unassisted.	Delay beyond 5th month.
6 mos.....	Recognizes immediate attendants.	Delay beyond 7th month.
7 mos.....	Lower central incisor teeth appear.	Delay beyond 8th month.
10 mos.....	Able to sit up.	Delay beyond 11th month.
12-13 mos.....	Talks. Says usual mono- and disyllables.	Delay beyond 14th month.
12-13 mos.....	Walks. Stands alone and takes few steps.	Delay beyond 14th month.
18 mos.....	Anterior fontanel completely closed.	Delay beyond 20th month.

Any delay in one or more of the usual physical advancements beyond the specified time should arouse suspicion of an accompanying mental retardation. Not all physical and mental retardation can be attributed to an endocrine disturbance. Other causes not infrequently found and which must be excluded are congenital lues, birth injury, intracranial hemorrhage, idiocy of varying types, neuromuscular dystrophies, cerebral palsy of childhood, amentia, moronism, microcephalus, macrocephalus, and the various types of spastic paralysis, malnutrition, poisoning (alcohol, lead, mercury) in the parent, early tuberculosis, chlorosis, leukemia, and the other hemic diseases.

In addition to a history of either definite or suggestive physical retardation and a study of the osseous system, there are

feet and around the wrists and ankles. The hands are broad and short, hence the term "spade hand." The cretin is

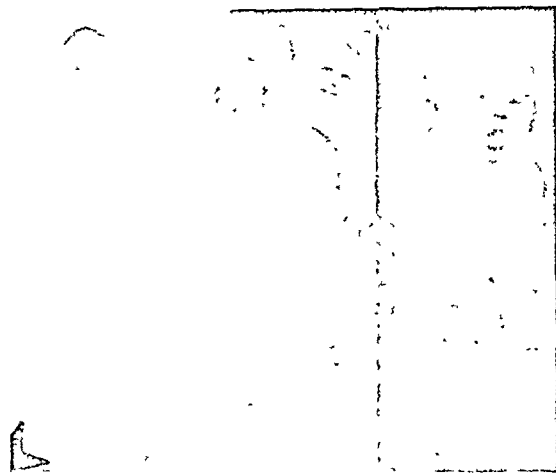


FIG. 2. Cretin with secondary hypophyseal disturbance shown at ages of one, seven, and eleven years. Note classical facies at one year of age.

certain physical findings in hypothyroidism of infancy. These findings correspond to, or are modifications of, those of the typical cretin. A typical cretin is of short stature and may or may not be fat. The skin is dry and frequently has a peculiar waxy tint. The hair is coarse and dry and there is a tendency to sparsity, often shown only in the eyebrows. Sutures and fontanels close late. The facial expression is characteristic, having a bloated appearance with a puffiness of the eyelids causing the eyes to appear small. Not infrequently there is a strabismus present and the bridge of the nose is flattened with the alae nasi thickened. The lips are thickened, the mouth is held open with a thickened tongue protruding. Drooling is a characteristic. Dentition is delayed with malocclusion common, and the teeth decalcify early. The protruding abdomen gives rise to the term "pot belly" and frequently an umbilical hernia is present. External genitalia in the male are small. There is lack of muscle tone with general muscular weakness which together with relaxation of the joints gives rise to a flaccid condition of the extremities. There are localized fat deposits over the dorsa of the hands and



FIG. 3. Case of thyroepituitarism at ages of eleven months, three, and ten years. Compare early photograph here with those in Figure 2.

gentle and affable, always ready to mimic. General mental retardation is plainly evidenced.

Figure 2 is that of a classical cretin showing him at different ages, one year, seven years, and eleven years. From the photograph taken at one year of age, the facial characteristics of a cretin are plainly evidenced. He is said to have weighed but 6 pounds at birth, first teeth erupted at sixteen months; he did not walk until two years of age and to date can say only disyllables.

Figure 3 is shown in comparison to Figure 2. Here is a patient closely approaching mutism associated with marked mental and physical retardation due to a primary hypothyroidism of congenital origin. He is the first and only child, cesarean delivery. Weight at birth was not recalled, though considered by the father to have been normal. Dentition occurred at six to seven months. He walked at about two years and three months, though very poorly, being weak in the knees. He said monosyllables at about twelve to fourteen months, but has never improved in this respect until the past year. He can now say only a few words. The characteristic features of the cretin are not detectable in

either of the two earlier photographs and it is in such a type of case that a history of delayed development and retarded osseous

percentage of cases. Such a goiter is termed adolescent or colloid and is the same type as that associated with pregnancy and

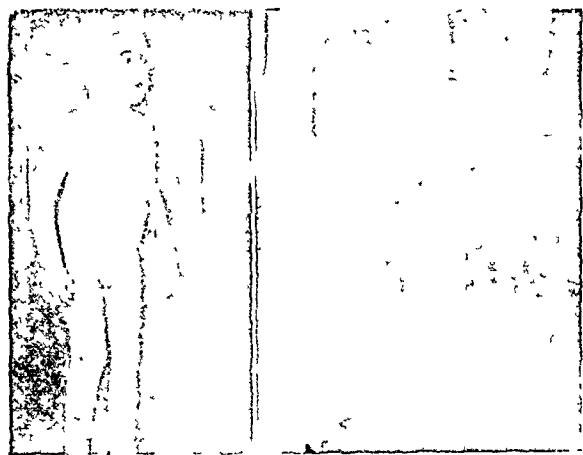


FIG. 4. Hypothyroidism in boy aged six years who has osseous development for child aged four years.

growth would be invaluable in making an early diagnosis.

Though rare, thyroid hyperplasia of the colloid type is occasionally seen in sporadic cretinism. Such a case is demonstrated in Figure 4. This boy, six years of age, could not talk, was incorrigible, nervous, and in general showed mental retardation. He weighed $9\frac{1}{2}$ pounds at birth, began to walk at about eighteen months, and said monosyllables at two years of age. His sense of speech has not improved since that time. A roentgenogram of the hand shows but four carpal centers present, while normally at this age there should be six.

In primary hypothyroidism in the female, early maturity frequently occurs. Figure 5A shows a girl ten years of age who began menstruating at the age of nine years and three months. Precocious secondary sexual characteristics are plainly evidenced. Like the other cases shown, this child also had a delayed developmental history.

During adolescence in children who apparently are normal in every respect, there frequently is noted a hyperplasia of the thyroid gland due to increased colloid formation. Basal metabolic rates are normal or below normal in the greater

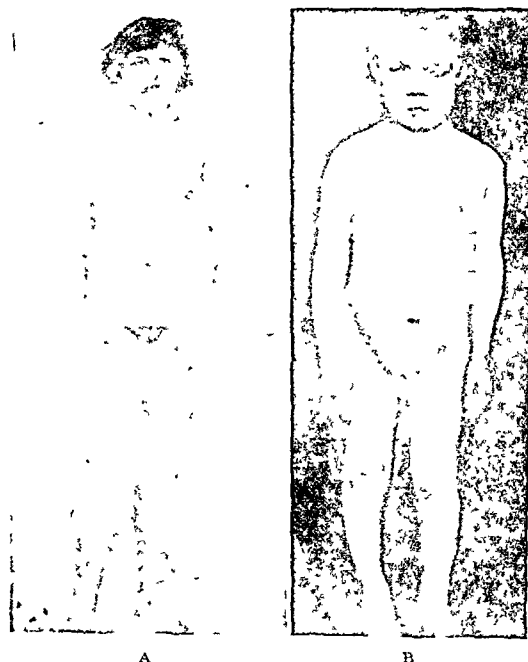


FIG. 5. Two types of cretins. A. Aged ten years, weight $128\frac{1}{2}$ lbs., height $58\frac{3}{4}$ in. (148.5 cm.), began menstruating at age nine years three months. B. Aged fifteen years, height $37\frac{3}{8}$ in. (94.8 cm.), has never had a menstrual period. (From Falta Myers.)

infections. In Figure 6 is shown a girl seventeen years of age with a colloid goiter which has been present since puberty.



FIG. 6. Adolescent goiter in girl seventeen years of age. Basal metabolic rate lowered. Mother of girl has had thyroidectomy for exophthalmic goiter.

This girl encountered considerable difficulty in her school work and was very shy, preferring solitude to companionship. Her

basal metabolic rate was -12 per cent. Her tolerant dosage of thyroid substance has been found to be 3 grains daily with marked improvement resulting. In the same figure is shown the mother of this girl who had been successfully operated on for an exophthalmic goiter occurring at the menopause. In the mother no history of long-standing goiter was elicited.

MYXEDEMA

Myxedema of the non-operative type is rarely seen before thirty years of age. It does occur occasionally in childhood, usually at the age of five to six years, "infantile myxedema." In such cases the characteristic adult myxedematous facies is found with the physical growth of a child.

Myxedema is the result of atrophy or disturbed function of the thyroid gland and is characterized by mental impairment, certain physical changes, subjective and objective, and almost always by a low basal metabolic rate. It is said to occur four out of five times in women. The onset is slow and the condition may exist for a long time before the patient seeks medical attention. Mental and physical signs usually run concurrently. At first, the patient may find himself satisfied to lounge around, leading an inactive life, disinterested in those things which previously had chiefly occupied his time. He becomes forgetful, prefers solitude or at least does not seek companionship. There is a gradual increase in weight with specific localization of fat paddings of dough-like consistency over the dorsa of the hands and feet, about the wrists and ankles, and over the supraclavicular and post-cervical areas and the face. There is a tendency for the eyes to appear small because of the encircling infiltrations about them, giving the face a puffy appearance. The skin becomes dry and thickened with a generalized non-edematous infiltration. The hair on the scalp, eyebrows, and eyelashes may become sparse and occasionally this is one of the earliest

signs. As the condition progresses, mental sluggishness becomes more marked, manifested by poor memory, somnolence, both nocturnal and diurnal, and a desire for solitude. Concentration is diminished, perception is reduced, and response is slow, due to combined mental and physical apathy. Such patients follow the line of least resistance, shirking responsibility at every turn. Acuity of the special senses is impaired and in marked cases dementia with hallucinations or delusions may result. This may progress to a true insanity with melancholia.

At a late stage, the lips, nose, and tongue are thickened, the voice is harsh, and the speech is slow and monotonous. Movements are clumsy and the patient fatigues easily. He complains of general body chilliness especially of the extremities with numbness and paresthesias of these parts. It is not uncommon for him to have general body myalgias, joint pains, anorexia and constipation. The pulse is usually slow though occasionally it may be moderately rapid, particularly in those cases where the patient has forced himself to keep going. The blood pressure, temperature, and basal metabolic rate are usually below normal.

Not infrequently there is an albuminuria found if the condition has existed for any length of time. A study of the renal system shows that there is only slight impairment of renal function and that the nitrogenous elements in the blood are usually within the normal or even reduced. Marked edema may be present which is readily reducible by thyroid therapy. Such a condition is commonly spoken of as "nephrosis."

There are various modifications of the above entity which, if coupled with a normal basal metabolic rate, cause one to hesitate in making a positive diagnosis. It should be borne in mind that occasionally the basal metabolic rate is normal in cases which are found to tolerate large doses of thyroid substance and improve when the therapeutic test is applied.

Figure 7 is that of a well-developed case of classical myxedema. The diagnosis on this patient was made some years

to be +12 per cent and an attempt to give him $\frac{1}{160}$ grain of thyroxin by mouth daily caused symptoms of thyrotoxicosis.



FIG. 7. Long-standing myxedema.

ago by Dr. William Englebach and the patient showed distinct improvement under treatment. Four months ago, she sought relief from her old symptoms of weakness, loss of memory, numbness over the entire body, paresthesias, somnolence, gastric distention, and a general feeling of chilliness over the entire body. She had not taken thyroid substance for the past three months. The basal metabolic rate at the time was -48 per cent. This patient readily improved on thyroid therapy.

Postoperative myxedema or cachexia strumipriva is seen occasionally even at the present time. Such a case is shown in Figure 8. This patient complained of lack of endurance, tachycardia, irritability, nervousness, and headache, all coming on with the slightest mental or physical exertion. He had had a thyroidectomy for toxic goiter ten months previously with onset of present symptoms a short time after the operation. The patient related that at three months' postoperative time, his basal metabolism was found



FIG. 8. Postoperative hypothyroidism, ten months after operation.

He reported for examination ten weeks ago at which time myxedema was suspected, though his basal metabolic rate was but -6 per cent on two occasions. Only small doses of thyroid substance were given (grain $\frac{3}{4}$ daily). Nine days later, the basal metabolic rate was -23 per cent and four days after this it was -15 per cent. The thyroid dosage was increased to tolerance which was found to be $4\frac{1}{4}$ grains daily, bringing about marked improvement in the patient's condition. Ten days ago the last observation was made at which time he reported himself feeling much better than he had for months. His basal metabolic rate was exactly normal and his thyroid tolerance had been reduced to $2\frac{1}{4}$ grains daily.

PRIMARY HYPOTHYROIDISM WITH SECONDARY INVOLVEMENT OF OTHER DUCTLESS GLANDS

Simple or uncomplicated hypothyroidism is relatively uncommon after

the age of five years, for at this time there usually is secondary involvement of either lobe of the hypophysis. Should

Figure 5 shows two types of cretins: (A) thyropituitarism with posterior lobe involvement and (B) thyropituitarism with

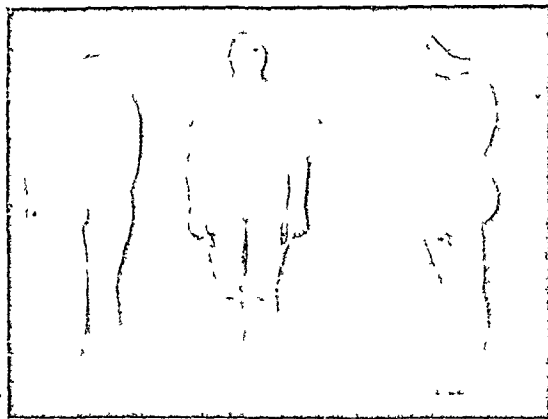


FIG. 9. Hypothyroidism secondary to primary pituitary dysfunction

the anterior lobe be involved, the statural growth is markedly retarded as is also the genital development. Should the posterior lobe alone be involved, statural

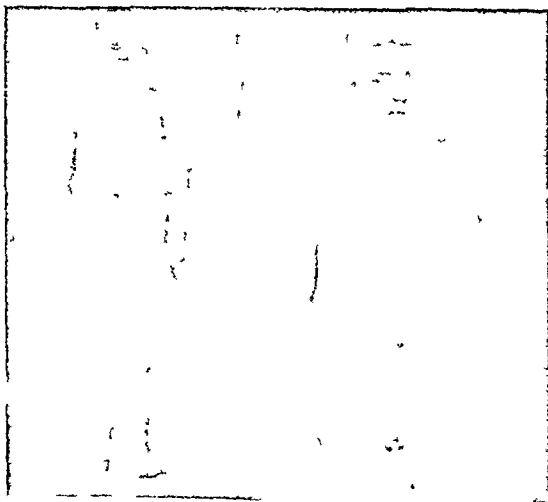


FIG. 10. Pituitary-thyroidism in male aged fourteen years. Note hypoplasia of genitalia and localized adiposity.

growth is not retarded, genital development is undisturbed or precocious, and "pituitary" type of obesity is present. Again, involvement of one lobe of the hypophysis will frequently, after a few years, bring about involvement of the other.

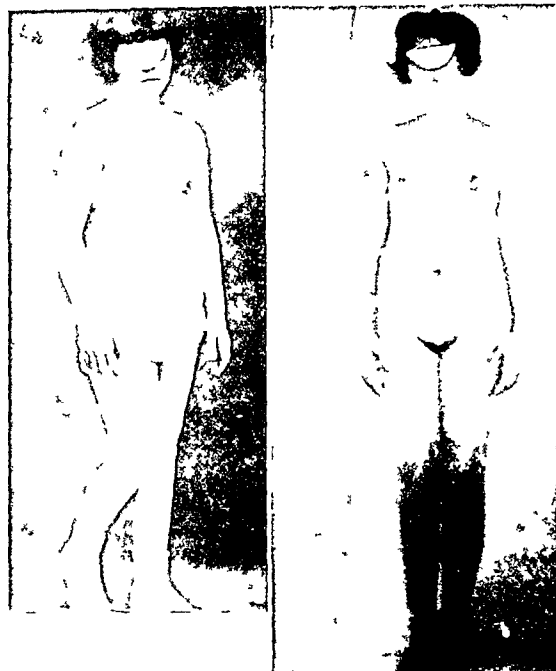


FIG. 11. Pituitary-thyroidism "pituitary, hibernation," in juvenile. Before and six months after treatment.

anterior lobe involvement. The patient shown in Figure 5 A, but ten years of age, was $58\frac{3}{4}$ inches (148.5 cm.) in height and had begun menstruating at the age of nine years and three months with regular periods thereafter. The patient in Figure 5 B, fifteen years of age, was $37\frac{3}{8}$ inches (94.8 cm.) tall and had never menstruated.

Just as there may be early maturity so may a late menopause and not uncommonly menorrhagia be associated with hypothyroidism.

HYPOTHYROIDISM SECONDARY TO INVOLVEMENT OF ONE OR MORE OTHER DUCTLESS GLAND DISTURBANCES

Clinically there is a very close relationship between the thyroid and other ductless glands. As has just been demonstrated, the pituitary frequently becomes involved secondarily when there is a primary hypothyroidism present. The antithesis to this may occur. Such a case is shown in Figure

9. From the history of normal mental and physical development until the age of seven years, when she began to gain weight

Another case is that of a girl aged sixteen years who suddenly became very somnolent, sleeping fourteen to fifteen

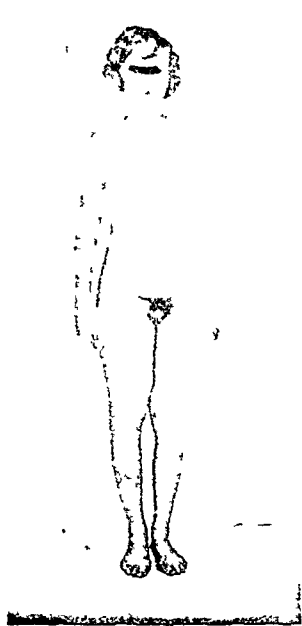


FIG. 12.

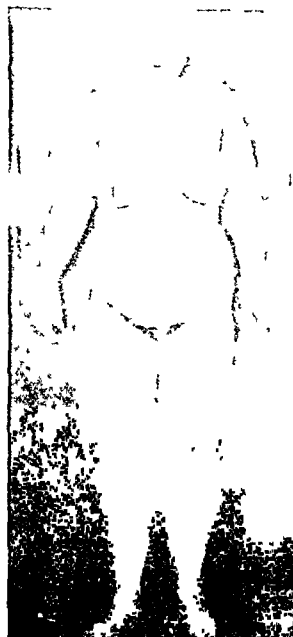


FIG. 13.

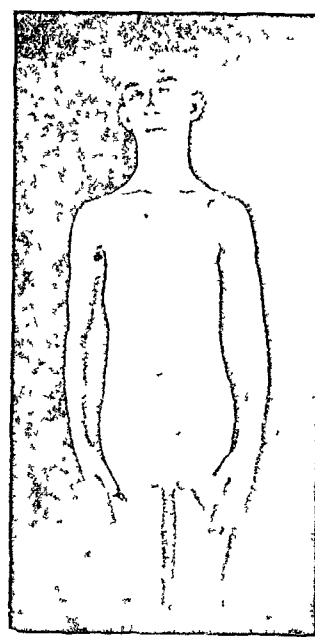
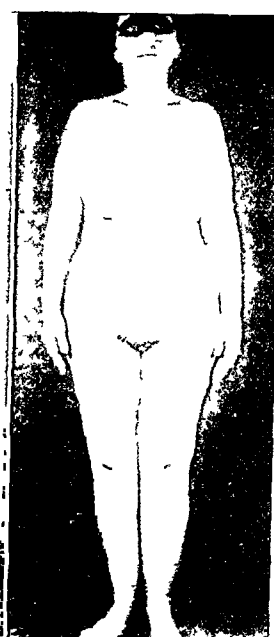


FIG. 14.

FIG. 12. Pituitary-thyroidism, "pituitary epilepsy." No attacks in eight months on combined pituitary and thyroid medication. Ketogenic diet added, which in itself on previous trial had been of no value.

FIG. 13. Pituitary-thyroidism in female aged thirty-eight years before and three months after treatment. Loss of 40 pounds.

FIG. 14. Hyperpnealism in boy who also had colloid goiter. Note precocious genital development.

excessively, and the absence of thyroid signs until just recently, it was determined that the posterior lobe of the pituitary was the primary glandular disorder. Engrafted on this was a secondary hypothyroidism.

Figure 10 shows a similar condition in the male in which there was involvement of both anterior and posterior lobes of the pituitary originally with a secondary thyroid dysfunction.

When the primary pituitary disturbance is marked, "pituitary hibernation" occurs. This is rare in a child and is seen only occasionally in adults. Figure 11 shows such a case before and after treatment. This patient was considered normal one year prior to time of observation when she was eleven years of age. She suddenly became very somnolent, lost interest in her school work and play, and became very irritable and dissatisfied. It was only with difficulty that she could be kept awake.

hours daily, and who had fallen asleep several times in the bathtub while bathing. These cases respond rapidly to combined pituitary and thyroid medication.

Figure 12 is a case of pituitary-thyroidism, anterior lobe involvement, with grand and petit mal. Just prior to the time of her examination, attacks of grand mal occurred at two weeks' intervals while petit mal attacks were numbering thirty to forty a day. This patient had previously been treated with luminal and a high fat diet, though strict ketonuria had not been maintained. She was of the anterior lobe pituitary type and her basal metabolic rate on two occasions was -15 per cent. She was placed on combined thyroid and pituitary treatment in addition to a strict ketogenic diet. She had one attack of grand mal the week following her first observation and has had none since, a period of eight and one-half months. Attacks of petit

mal now occur only occasionally and are barely detectable.

In middle adult life, patients with combined pituitary and thyroid disturbance assume such proportions as demonstrated in Figure 13. This patient, thirty-eight years of age, weighed 218½ pounds when she first came under observation and in her complaints were included many nervous symptoms. Exactly four weeks later, she weighed 199¾ pounds, a loss of eighteen and three-quarters pounds, and there was distinct improvement in her nervous symptoms. Combined pituitary and thyroid therapy was administered.

Figure 14 shows a patient with a primary pineal disturbance in a boy aged fourteen years. Precocious genital development is evidenced. This boy had a goiter of moderate size which subsided almost entirely on thyroid substance.

PROGNOSIS

The prognosis in congenital or infantile hypothyroidism depends entirely upon the degree of hypofunction and the time that treatment is instituted. Treatment instituted late will result in improvement directly proportional to the time it is started and the intensity of its administration.

In myxedema and hypothyroidism of mild degree, the response to thyroid therapy is excellent. In cases having other glandular involvement, the best results with thyroid therapy are only obtained by treating the other glandular dyscrasias in conjunction.

TREATMENT

In all cases, the best effect of thyroid therapy is obtained only by giving the tolerant dosage. In infancy, this is measured by carefully watching the rectal temperature. An overdosage of thyroid substance will cause the rectal temperature to increase over 100°F. In juveniles, the tolerant dosage is measured best by

the outward manifestations. The dosage at this age should be maintained at a level just below that point which causes nervousness, irritability, restlessness, insomnia, tachycardia (pulse rate above 100), etc. In the adult, the thyroid dosage can in most cases be regulated best by basal metabolism.

CONCLUSIONS

1. It may be possible to prevent congenital cretinism by determining the basal metabolic rate in a pregnant woman before and after the fifth month of pregnancy. month of pregnancy. If it does not show an increase approaching 20 per cent over what it was during the first months of conception, thyroid medication is indicated.
2. Diagnosis of cretinism during the first few months of life is made from the history of delayed physical development, not due to other causes, retarded osseous development, and physical findings.
3. After the fifth year of life, there is usually found associated with primary hypothyroidism a disturbance of one or more of the other ductless glands. The hypophysis is most commonly involved. Hypothyroidism may occur either primary or secondary to other glandular dysfunction.
4. Occasionally a normal basal metabolic rate is found repeatedly in patients who improve on relatively large doses of thyroid substance showing that a state of hypofunction had existed.
5. In early hypothyroidism, prognosis depends upon the time treatment is instituted and the intensity of its administration.

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CARDIAC IRREGULARITIES ASSOCIATED WITH DISEASES OF THE THYROID GLAND*

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THIS discussion will be confined to those irregularities or disturbances in the mechanism of the heart beat so frequently found associated with thyrotoxicosis. The irregularities found in that group of young adults living in the region of the Great Lakes who show simple colloid enlargement of the thyroid gland are probably due to some other cause and have no connection with the glandular enlargement.

A number of excellent papers have recently been published on the changes in the heart associated with hypothyroidism and myxedema. Hypertension, nephritis and arteriosclerosis are so frequently associated with this disease, that it would be difficult or impossible to determine which factor was directly responsible for any disturbances in the mechanism of the heart beat which might be present.

Patients with toxic goiter, whether of the exophthalmic type or those showing adenomatous degenerative changes with hyperthyroidism, usually have a definite increase in the resting heart rate. This sinus tachycardia practically eliminates from our discussion two of the most frequently encountered cardiac irregularities: sinus arrhythmia and premature contractions. Sinus arrhythmia is almost always found when the heart is beating slowly and tends to disappear as the heart rate increases. Premature contractions, or the so-called extrasystolic type of arrhythmias, usually disappear promptly with an increase in the heart rate. Premature contractions are unusual with a ventricular rate above 120.

We have seen but one case of paroxysmal auricular tachycardia associated with toxic goiter. This occurred in a young woman aged twenty-two who gave a history of

having had similar attacks since early childhood. They had their onset following a severe attack of scarlet fever when she was eleven years old. She had numerous attacks while being prepared for operation and they continued to occur postoperatively, but at less frequent intervals, and the attacks were of shorter duration. While the condition may have been aggravated by the presence of the thyrotoxicosis we were of the opinion that the two were not directly related.

Undoubtedly the most frequently encountered disturbance in the mechanism of the heart beat in thyrotoxicosis is auricular fibrillation. It was present at some time in the course of the disease in 23 per cent of our cases. Auricular flutter was much less frequent but since both are due to a circus rhythm and since the changes in the myocardium which permit their presence are probably identical, we have grouped them together and they will be discussed as possessing similar significance.

We have divided our cases showing thyrotoxicosis and auricular fibrillation or flutter into four groups:

I. Those showing paroxysmal fibrillation or flutter very early in the course of the disease and before any other manifestations of thyrotoxicosis were detectable.

II. Those showing paroxysmal fibrillation or flutter early in the course of the disease but in which other signs of toxicity were evident.

III. Those showing permanently established fibrillation or flutter and in which toxic symptoms were easily distinguishable.

IV. Those in which fibrillation or flutter did not appear until after operation and then as a part of the so-called thyroid shock or crisis with a very rapid ventric-

* Read at the Annual Meeting of the American Association for the Study of Goiter, Dayton, March 25-27, 1929.

ular rate and other signs of profound intoxication.

This discussion will be restricted to those cases which fall into group 1. To date we have studied 6 patients who showed auricular fibrillation or flutter so early in the course of the disease that careful repeated physical and laboratory examinations failed to show any of the other signs of thyrotoxicosis for weeks or months following the first paroxysm. The history and course of the individual cases are so similar that we have chosen one case for discussion which is typical of the group.

Miss L. K., a twenty-eight-year-old accountant, was referred to us with a tentative diagnosis of paroxysmal auricular tachycardia. The patient stated that with the exception of measles and whooping cough at five years of age she could not remember of having had a day's illness in her lifetime. Three weeks before she came to us, while exerting herself rather violently in a gymnasium, the heart rate suddenly became very rapid, she became markedly dyspneic, her face and body were covered with a cold perspiration, and she felt very weak, exhausted and nauseated. She was assisted to the dressing room and her family physician was called. It was about twenty minutes before he could reach her and in the interim the attack ceased as suddenly as it began, following which the patient felt perfectly well. Her physician made a diagnosis of "nervous heart" and suggested that she stop her gymnasium work for a time. Golf was substituted for exercise in the gymnasium and for the next three weeks she enjoyed her usual good health. At the end of the third week while walking rapidly up a hill she had another attack. She said that her heart rate seemed much faster than during the first seizure, and in addition to her previous symptoms she vomited. She was immediately taken home where she was met by her family physician, who had an opportunity to observe the character of the attack. He made a diagnosis of paroxysmal tachycardia and referred the patient to me for further study.

Physical examination showed a well-developed, well-nourished young woman with excellent color and apparently in the best of health. The tonsils were moderately enlarged and some pus could be expressed from the

crypts. Roentgen-ray examination of the teeth showed an apical abscess of the first molar in the right lower jaw.

Examination of the eyes showed no exophthalmos or lid-lag. The lobes of the thyroid were palpable with difficulty, but swallowing revealed some slight enlargement in the region of the isthmus. With the arms extended and the fingers widely spread there was no trace of fine tremor. She was very calm during the examination and while the skin over the body was soft and moist there was no abnormal perspiration. When questioned about nervousness the patient said that the examination did not disturb her at all and that she had never been nervous in her life. She said that she perspired only on strenuous exertion. Examination of the heart showed little of significance. The rate at the apex was 80 per minute; the rhythm was regular, with no evidence of overactivity. The heart was not enlarged, the left border being in the fifth intercostal space well within the mid-clavicular line. Auscultation at the apex revealed a very soft, distant, blowing systolic murmur but no diastolic murmurs were audible either at the apex or at the base. The examination of the lungs was negative. The remainder of her physical examination showed nothing significant. The blood pressure was 120/70. The urine was normal. A teleoroentgenogram showed the heart to be small, normal in shape, with no change in the vessels at the base. The Danzer ratio was 0.43. The lung fields were negative. An electrocardiographic tracing showed a ventricular rate of 80 per minute, there was no sign of fine muscle tremor in the tracing and no preponderance of either side of the heart. The conduction time was normal. The blood Wassermann and Kahn reactions were negative. Examination of the blood showed nothing abnormal. A basal metabolic rate was not done at this time.

Since the patient revealed no abnormalities except infected tonsils and one dental abscess, and not having seen her in an attack, I made a provisional diagnosis of paroxysmal auricular tachycardia. The patient was told to have her tonsils and the abscessed tooth removed at once and if she had any more attacks to come in for an electrocardiographic tracing.

There was no change in her condition for nine weeks after the removal of the tonsils and tooth. One evening after hurriedly going up stairs she was seized with another severe

paroxysm of tachycardia. She was brought to the hospital at once in an ambulance. An electrocardiographic tracing was taken, which showed auricular fibrillation with a very rapid ventricular response of 180 per minute. She was very uncomfortable and was given morphine sulphate grains $\frac{1}{4}$ and 10 c.c. of a proprietary digitalis preparation intravenously which had an approximate cat unit strength of one cat unit per cubic centimeter. Two hours later the ventricular rate had fallen to 110 per minute. The attack persisted, however, until four o'clock the following afternoon. At this time the patient was again carefully questioned regarding any previous manifestations of rheumatic infection and the heart was re-examined for mitral or aortic disease. Our findings did not justify such a diagnosis. For three successive days following cessation of the attack basal metabolic determinations were made. She cooperated well and the tests were considered entirely satisfactory. The rates were plus 12, plus 14 and plus 16, an average of plus 14. The patient was again re-examined in the hope of finding some evidence of thyrotoxicosis. Our findings were practically identical with those found on previous examination and while we were very suspicious of this condition, we did not feel justified in making such a diagnosis. She was discharged with instructions to keep us informed as to subsequent attacks and to keep careful watch for nervousness, tachycardia, sweating, etc.

She had four more attacks of fibrillation during the next seven months. Electrocardiographic tracings were obtained during all of these attacks which lasted from one hour to two days. Basal metabolic rates were taken following each attack and the highest reading obtained was plus 20 just after the last attack. The patient was also re-examined following each paroxysm. Nothing was found on clinical examination to justify a diagnosis of either rheumatic heart disease or thyrotoxicosis.

Three weeks after the last attack she came to my office complaining of increasing nervousness with a desire to be constantly on the move. She also complained of moderate tachycardia, profuse sweating, particularly at night, insomnia and some slight dyspnea on exertion. Her physical examination at this time showed a sinus tachycardia of 110; the heart was definitely overactive; there was now a distinct fine tremor of the fingers; the blood pressure

was 135/90 and the skin was covered with fine perspiration. The basal metabolic rate was plus 40. The electrocardiogram now showed a sinus tachycardia with fine muscle tremor quite marked in all three leads. As yet there was no change in the eyes. A diagnosis of Graves' disease was made and the patient admitted to the hospital. She was given the usual preoperative treatment including Lugol's iodine minims 5, three times daily. By the end of the second week her basal metabolic rate had fallen to plus 20, and most of the nervousness, sweating and tachycardia had disappeared. A thyroidectomy was done on the fifteenth day. Her progress following operation was uneventful and she was allowed to go home at the end of the third week. Microscopic examination of the tissue removed showed the typical epithelial hypertrophy and hyperplasia and lymphoid hyperplasia of Graves' disease. This patient has been observed for over a year following operation. Her general health has been excellent and there has been no return of the fibrillation.

DISCUSSION

Cases of this type raise two questions in my mind. First, should auricular fibrillation or flutter be considered one of the earliest manifestations of thyrotoxicosis in certain cases; second, are the changes in the myocardium in this disease which permit the onset of circus rhythm always indicative of widespread and permanent structural changes in the heart muscle.

To answer question one in the affirmative without explanation or reservation would undoubtedly lead to misunderstanding and criticism. We know that to find auricular fibrillation in thyrotoxicosis long before there is a detectable change in the basal metabolic rate or other clinical manifestations of the disease is the exception rather than the rule. However, the case we have discussed is only one of 6 which we have collected and serves to emphasize that in certain cases fibrillation may be the earliest manifestation of the disease. If we keep this point constantly in mind a great many more such cases would undoubtedly be found.

Question two is very difficult to answer,

and we are doubtful if an accurate opinion could be rendered. We know little of the nature of the myocardial changes produced by thyroid intoxication. Whether the changes in the myocardium which permit the onset of the circus rhythm are structural, physicochemical, or a combination of both is not clear. We feel that permanently established auricular fibrillation is probably associated with more or less permanent damage to the heart muscle, since fibrillation usually recurs in this type of case after being abolished by quinidine. Pale muscle fibers with indistinct striations, lipoidal changes, and localized necroses have been described as changes in the heart muscle secondary to toxic thyroid disease. Whether these changes are specific for this disease has certainly not been established.

The mechanism of the production of transient or paroxysmal fibrillation in the type of cases described above is probably due to temporary changes in the myo-

cardium. The fact that the paroxysms of fibrillation cease following operation indicates that the myocardium probably recovers more or less completely from whatever injury may have occurred. This viewpoint is also supported by the absence of subsequent cardiac enlargement, changes in the T-wave in the electrocardiogram or the development of signs of cardiac weakness or failure.

CONCLUSIONS

1. Auricular fibrillation or flutter are the most frequently encountered irregularities or disturbances in the mechanism in the heart beat associated with thyrotoxicosis.
2. Auricular fibrillation or flutter may be the earliest detectable sign of thyrotoxicosis.
3. The nature of the changes in the myocardium which accompany the development of early paroxysmal fibrillation has not been established, but they are apparently transient.



OCCURRENCE OF ASTHMA IN PATIENTS MANIFESTING EVIDENCES OF THYROID DYSFUNCTION*

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CHICAGO

IN certain respects asthma and hyperthyroidism seem to stand in opposition to each other. *Asthma* is thought to be produced by peripheral or central stimulation of the parasympathetic nerves causing spasm of the constrictor muscles of the bronchi. It is relieved temporarily by adrenalin. *Hyperthyroidism* is accompanied by many manifestations suggesting overstimulation of the sympathetic nervous system: tachycardia, vasomotor dilatation, exophthalmos, etc. Many of these manifestations are aggravated by adrenalin. The occurrence of asthma in hyperthyroid states is, consequently, hardly to be expected. Since such an association of clinical phenomena is occasionally observed, and if this relationship is anything more than casual, a satisfactory explanation of the physiological processes involved should be of considerable interest.

A survey of the literature discloses the fact that American physicians have not written upon this subject and but scant mention of it is made in European publications, although Widal and Abrami¹ and Danielopolu² have written most interestingly upon it. Widal and Abrami have noted the association of asthma in both hyperthyroid and hypothyroid states. In one patient in whom asthma and hyperthyroidism were associated attacks of asthma could be induced at will by the administration of thyroid extract. Danielopolu maintains that evidence of both sympathetic and parasympathetic overstimulation can be detected in all cases of

exophthalmic goiter and uses the term "amphotony" to designate the overstimulation of these opposing nerve groups. The resulting effect, i.e. whether sympathotonic or vagotonic, varies in individual patients according to which group is dominant. This is in opposition to the view of Eppinger and Hess that cases of hyperthyroidism may be divided into two distinct groups, the one showing symptoms produced only by overstimulation of the sympathetic system (sympathotonic), the other showing evidence of overstimulation of the vagus (vagotonic or vegetative).

In view of the above, observations on 6 patients who suffered from typical attacks of asthma and who also had manifest disturbance of thyroid function may not be without interest and are herewith given:

CASE I. Mrs. B. D., a Jewish housewife under observation since September 1920, at which time she was thirty years of age. She is said to have always been emotionally unstable, to have had a goiter since fifteen years of age, and to have been a sufferer from paroxysmal asthma which seemed to follow a severe respiratory tract infection since the age of twenty-six. She has had chronic bronchitis since the onset of the asthma. She was overweight, had definite oral and tonsillar infection (later eradicated), and was slightly sensitive to a few proteins, most markedly so to horse serum (++++). The paroxysms of asthma were relieved by inhaling the fumes of burning asthma powder and by adrenalin, but were apparently not benefited by the removal of focal infections nor by intravenous injections of peptone. Her thyroid status was considered normal until the fall of 1925; then the goiter

¹Widal, F. and Abrami, P. *Presse méd.*, 32: 473-476, 1924.

²Danielopolu, D. *Bull. et mém. Soc. méd. de hôp. de Par.*, 49: 234-239, 1925.

* From the Medical Department, Northwestern University Medical School. Read at the Annual Meeting of the American Association for the Study of Goiter, Dayton, March 25-27, 1929.

became larger, she was more nervous and mentally unstable, and she complained of palpitation. The basal metabolic rate was +27 per cent. Hyperthyroidism was diagnosed, iodine was given, following which she became more nervous and excitable, although the basal metabolic rate fell to +17 per cent.

Subtotal thyroidectomy (Dr. Allen B. Kanavel) was performed on February 18, 1926; a large nodular and partially substernal goiter was removed. Basal metabolic rate dropped to -8 per cent. She left the hospital apparently convalescing satisfactorily from the operation but returned one month later mentally unbalanced with manifestations resembling the manic stage of a manic-depressive psychosis; she had insomnia and was greatly depressed although she would laugh easily and long on slight provocation. The psychosis cleared up in six weeks. The basal metabolic rate remained normal and, although emotionally unstable, she seemed in a normal thyroid status. There have been no further attacks of asthma and she has had but little bronchitis since the thyroidectomy.

Of interest are the observations that this woman, who was always emotionally unstable, who had had an apparently simple goiter since the age of fifteen, became asthmatic following a respiratory infection at twenty-six, became hyperthyroid at thirty-five, developed a severe psychosis following a "cold" while convalescing from a thyroidectomy in 1926, and the asthma has apparently disappeared although a tendency to bronchitis persists, since the operation.

CASE II. Mrs. T. H. C., a housewife of sixty years, under observation since December 18, 1926. She is said to have had seasonal (March to May) and paroxysmal asthma, first at the age of nineteen but not again until twenty-seven, although she has been asthmatic each spring since twenty-seven. Thyroid enlargement was first noted at the age of thirty-nine and has persisted to the present time. The thyroid enlarges in the spring of the year and recedes in the autumn. She has taken 10 grains of potassium iodide each night while suffering from asthma with considerable relief and gets complete although temporary relief from severe attacks of asthma by adrenalin subcutaneously administered. Aside from the asthma she has noted hoarseness, weakness, heart consciousness, puffiness of the face, dryness of the skin, and occasionally nausea

and vomiting. All symptoms, including the asthma, were worse during the months when the thyroid was enlarged.

Examination showed the skin dry, the face puffy and wrinkled, the thyroid enlarged and nodular, the chest emphysematous, respiratory sounds asthmatic and wheezing; roentgen ray of the chest was negative although a questionable shadow suggesting substernal goiter was present; the heart was not enlarged, rate 72, with occasional preventricular contractions; blood pressure 102-62; nasal polypi with infection and oral infection about two teeth were present. Protein sensitization tests were negative. Basal metabolic rate: -12 per cent. She was evidently in a hypothyroid state although myxedema was not definitely diagnosed.

On thyroid extract the basal metabolic rate was elevated to +13 per cent, the thyroid receded in size; the asthmatic signs became much more severe. Thyroid feeding was stopped; basal metabolic rate dropped to -26 per cent and the thyroid again enlarged. The patient complained of pain in the arms, sleepiness, nausea and vomiting. Thyroid feeding was again tried and the asthma again became much more severe, the heart became irregular; weakness of the hands and atrophy of muscle groups in the forearm and hands (interossei, thenar and hypothenar eminences) were observed. Thyroid feeding was again discontinued.

On no medication the patient is said to have improved gradually although she has not been seen by me since February 5, 1927. She is now in California.

This patient is of interest because asthma and nodular goiter have been present for many years, the asthma being worse during periods of enlargement of the thyroid gland. She is definitely hypothyroid; the asthma is benefited by iodine and adrenalin and made worse by thyroid feeding. Muscle atrophy, apparently due to neuritis, and gastrointestinal disturbances are associated.

CASE III. Miss G. B., a dietitian, thirty-five years of age, previously healthy, contracted an acute respiratory tract infection with fever and chills in October, 1926. Five days thereafter she experienced the first of many, almost daily, severe paroxysms of asthma associated with orthopnea and cyanosis. Chronic bronchitis developed and continued thereafter.

Physical examination revealed little save asthma. The thyroid was not enlarged; radiograms of the chest were negative; roentgenograms of the sinuses showed cloudiness of both maxillaries but washings of sinuses gave negative results. She was found sensitive to horse serum but less so to other proteins. The acute attacks of asthma, orthopnea and cyanosis were relieved by both adrenalin subcutaneously and ephedrin by mouth.

She was sent to Colorado but found no relief there from the asthma; in addition palpitation, weakness and loss of weight ensued. She then went to Arizona where some relief from the asthma was experienced but heart consciousness, palpitation, dyspnea, weakness, loss of weight, flushing, tremor and thyroid enlargement were noted. Basal metabolic rate +24 per cent. Blood pressure 90-67.

In May, 1928, she re-entered the hospital. Asthmatic attacks were severe and frequent, the thyroid was slightly enlarged, the heart normal, the pulse 100; she was nervous, tremulous, apprehensive. Basal metabolic rate +18 per cent. A diagnosis of hyperthyroidism was made. Lugol's solution (gm. 15 t.i.d.) was given. The asthma improved, the thyroid became larger, and palpitation, dyspnea, restlessness, sleeplessness and instability increased.

On May 28, 1928, a subtotal thyroidectomy was performed (Dr. A. L. Shreffler); an adenomatous goiter and a large accessory thyroid gland were removed. Postoperative recovery was slow but she gradually improved and gained weight and strength. Basal metabolic rate -18 per cent.

She contracted a severe acute respiratory tract infection, then epidemic, in October, 1928. She became very ill; fever, dyspnea and cyanosis were present; pneumonia was suspected but not proved. Convalescence was slow. On December 3, 1928, she was readmitted to the hospital and acute purulent infection of both maxillary sinuses was found. Drainage of the infected sinuses was established. She recovered promptly and now seems quite well, has regained her strength and weight, and has had no further attacks of asthma.

Of interest in this case is the apparent relationship of both asthma and hyperthyroidism to both an acute and chronic respiratory tract infection; also the apparent recovery following thyroidectomy and the subsequent drainage of the maxillary sinuses.

CASE IV. Mr. J. E. W., a secretary, fifty-three years of age, who had always enjoyed good health, in February, 1928, began having paroxysms of asthma without apparent cause. The attacks are severe, mostly nocturnal, and are associated with dyspnea and orthopnea. He is dyspneic on exertion. On the advice of a physician, eighteen teeth were extracted. Thereafter mastication was difficult on account of insufficient masticating surface; asthma continued; he lost 22 pounds in weight.

Examination on July 17, 1928, showed the thyroid palpable, tremor present, chest emphysematous, breathing asthmatic, the heart not enlarged, tones distant, pulse 144. Blood pressure 160-90. Roentgenograms of the chest showed a substernal goiter with pressure on the trachea, lung markings as though of bronchitis, pleural adhesions and a small heart. Basal metabolic rate +50 per cent. He was found sensitive to corn, clover and silk. Asthmatic attacks were temporarily relieved by adrenalin and ephedrin. Diagnosis: asthma, hyperthyroidism (substernal goiter), oral infection (eradicated), and hypertension. On iodine but slight benefit was noted although the basal metabolic rate dropped to +30 per cent.

A subtotal thyroidectomy was performed on August 21, 1928 (Dr. H. M. Richter). But little improvement has followed the operation although his basal metabolic rate is +6 per cent. He had some symptoms of postoperative tetany of light grade, blood calcium 9.5 mg. Tachycardia has disappeared; hypertension is present; he has gained 23 pounds in weight. He states that he feels stronger but the asthma has continued, the attacks being possibly less severe, in spite of an apparently normal thyroid status having been established.

Of interest in this patient is the development of severe hyperthyroidism four months following the onset of asthma in a man who has severe nephritis with hypertension. The asthma has not been relieved by the establishment of a normal thyroid status by thyroidectomy.

CASE V. Mrs. M. F., a Jewish housewife, forty-five years of age, has had "hay-fever" in June and July each year since 1917. The first attack is said to have followed an acute respiratory tract infection. In January, 1927, weakness, nervousness, loss of weight, tachycardia and palpitation were noted; basal metabolic rate +35 per cent. Diagnosis: hay-fever, hyperthyroidism and chronic tonsillitis. Iodine

was administered with little effect on the basal metabolic rate. Subtotal thyroidectomy was performed on April 8, 1927 (Dr. H. M. Richter). Three severe attacks of asthma had occurred prior to operation while the patient was definitely hyperthyroid, and five days following the operation she again began experiencing frequent and severe paroxysms of asthma while definitely hypothyroid (postoperative myxedema) with basal metabolic rate -30 per cent. The basal metabolic rate was raised to normal by thyroid feeding but the asthma continued; the severe attacks were relieved by hypodermics of adrenalin.

On April 10, 1928, i.e. one year following thyroidectomy and while taking 5 grains of desiccated thyroid daily, exophthalmos, not previously noted, was present in the left eye. This receded on iodine but increased later while feeding desiccated thyroid sufficient to maintain the basal metabolic rate at normal. The patient has had less asthma during the past winter when requiring $7\frac{1}{2}$ grains of desiccated thyroid to maintain the basal metabolic rate at normal. In June, 1929, latent bilateral maxillary sinusitis was recognized and adequate drainage was effected by operative measures.

This case is of interest because hyperthyroidism developed in a patient who had had "hay-fever" for years. Severe paroxysmal asthma first occurred following a respiratory tract infection while the patient was presumably in normal thyroid status, continued later while she was hyperthyroid, and persisted still later while hypothyroid (postoperative myxedema). The severe attacks of asthma were relieved by adrenalin before (while hyperthyroid) as well as after thyroidectomy, at which time the basal metabolic rate was maintained at normal by thyroid feeding. Chronic maxillary sinusitis may have been a factor in producing both the asthma and hyperthyroidism. It is too early to estimate the effect that permanent drainage of the maxillary sinuses may have on her disability. Exophthalmos developed one year after thyroidectomy at a time when the basal metabolic rate was being maintained at normal by thyroid feeding.

CASE VI. Mrs. M. LaP., an Italian woman of forty-three years, gave birth to her ninth baby on November 5, 1928. The labor was normal and uneventful. She is said to have

had frequent colds and to have had known nephritis with hypertension for three years. She was first seen on January 16, 1929, on account of severe, mostly nocturnal, asthma of five days duration.

On examination she was found nervous, tremulous, the skin flushed and moist; the thyroid was palpable; the chest was emphysematous with many râles and asthmatic breathing; the heart showed left hypertrophy, a systolic murmur at the apex and over the base; pulse 110, regular; blood pressure 218-126. The eye grounds showed marked vessel changes of the nephritic type; the urine contained albumin and casts. Roentgenograms of the chest showed pulmonary congestion, enlargement of the heart and aorta, and a substernal goiter. Basal metabolic rate +29 per cent; January 28, +34 per cent; January 31, +52 per cent. Diagnosis: recent pregnancy, chronic nephritis with hypertension, cardiac asthma, substernal goiter and hyperthyroidism.

She was confined to bed and given Lugol's solution (gm. 15 t.i.d.) for sixteen days. Her general condition improved. Basal metabolic rate February 14, +22 per cent; February 23, +37 per cent. On account of the extensive pathology which was manifest, thyroidectomy was withheld and she was allowed to go home and was instructed to continue iodine under observation as an ambulatory patient.

Of interest in this patient is the fact that she has had known nephritis with hypertension for at least three years, probably aggravated by frequent pregnancies. She has a substernal goiter, is probably now hyperthyroid, and the asthma as well as the hyperthyroidism has apparently been influenced by bed rest and iodine. Should her condition become worse while on ambulatory regime thyroidectomy should be considered.

SUMMARY

Six patients who had asthma but who also had manifest disturbance of thyroid function are described. The clinical picture as to the thyroid status was atypical in all. There is a definite history, or evidence on examination, of a general infection or of gross focal infection, associated in 5 of these patients. In 2 the asthma has ultimately disappeared on establishing a normal thyroid status and eliminating focal infection. Hypertension with nephri-

tis was present in 2 of these patients and may have been a factor in the production of asthma in both. A substernal goiter, none large, was present in 3 of the patients operated upon; in only 1 was it of sufficient size so that pressure might have been considered a factor in the production of asthma. Four of these patients were definitely sensitive to foreign proteins.

One patient was hypothyroid throughout the period of observation; 5 were considered hyperthyroid. On 4 of the latter a subtotal thyroidectomy was performed and 1 of these developed postoperative myxedema. Paroxysms of asthma were observed in 5 while in a hyperthyroid state, and in 2 while definitely hypothyroid.

Adrenalin relieved 4 temporarily of asthma while hyperthyroid, although 1 had a frightful general reaction. Adrenalin relieved 2 of asthmatic attacks during periods in which they were definitely hypothyroid.

Iodine had a beneficial effect on the

asthma in 1 patient who was continuously hypothyroid, but no conclusion can be drawn as to the effect of the iodine alone on the asthma of the others although it was given to all of them, and the thyroid status of 4, while definitely hyperthyroid, seemed improved thereby.

CONCLUSIONS

No conclusions are drawn. The clinical observations here reported suggest, but do not demonstrate, that a physiological relationship may exist between asthma and hyperthyroidism. Their coexistence may be merely casual. The fact that but 6 patients thus afflicted have been seen in this clinic during the past eleven years would tend to support the latter view. Since 2 of these patients have been definitely relieved of asthma and hyperthyroidism by thyroidectomy and clearing up of definite focal infections, such a procedure seems warranted as a method of treatment.



THE EFFECT OF IODINE AND THYROID FEEDING ON THE HISTOLOGY OF THE GLAND OF THE DOG*

FURTHER STUDIES

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ABOUT a year ago we studied the histological effect of the administration of iodine and thyroid extract to the adult dog.¹ From these experiments we were led to agree with the conclusions of Marine,² that iodine stimulated the cells of the thyroid acini to produce colloid. Colloid was apparently secreted in large quantities, to such an extent that mechanical compression of the cells occurred. The effects of iodine and thyroid substance were apparently not distinguishable. Frequently the administration of iodine or of iodine and thyroid substance produced the histological picture of thyroid exhaustion.

Because of certain variations in the histological picture produced after similar rotations of feedings it was decided to reproduce the experiment under better methods of control.

The purpose of the present experiment was to observe the histological effect in three groups of animals. In the first group after biopsy the dogs were given iodine in the form of Lugol's solution, minims 20, daily for twelve weeks, when a second biopsy was done. After a rest period of eight weeks another biopsy was done. Iodine was then again administered for twelve weeks. After biopsy a second rest period of eight weeks was given, after which a final biopsy was done.

In the second group thyroid substance replaced the iodine for the second ingestion and was administered for twelve weeks in sufficient quantity to produce the symptoms of hyperthyroidism in the animals. No attempt was made to measure the basal metabolism.

In the third group one administration of iodine for twelve weeks was followed by an indefinite rest period of thirty weeks.

In order not to confuse the microscopic picture, the sections were removed from first one lobe and then the other, and from opposite poles when the lobe was operated subsequently. This we felt would permit of sections for study in which the trauma of a previous operation could possibly be avoided.

HISTOLOGICAL STUDIES

The microscopic pictures of the sections removed from the normal animals after being fed the usual animal house diet for several weeks were practically the same. The acini varied in size and in the amount of colloid they contained. The cells lining the acini were of a low cuboidal type with large dark-staining nuclei. There were here and there between the acini a moderate number of epithelial cells. (Figs. 1 and 2.)

The typical iodine effect was obtained in every animal after iodine feeding. The acini became distended with colloid. The cells were flattened and elongated. The cytoplasm seemed to have disappeared to a large extent and the compressed cells or nuclei appeared to form a limiting membrane from the adjacent acinus. There was a marked reduction in the number of interstitial cells. (Fig. 3.)

After the first rest period the histologic picture varied. In some sections the cells had regained a thin cuboidal appearance and the amount of intra-acinar colloid had diminished. In other sections the iodine effect appeared to have persisted. After the second period of iodine feeding

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we did not observe the same histological effect as occurred after the first twelve weeks of iodine feeding. The acini were

the grandular structure were returning toward the normal.

After the second rest period following

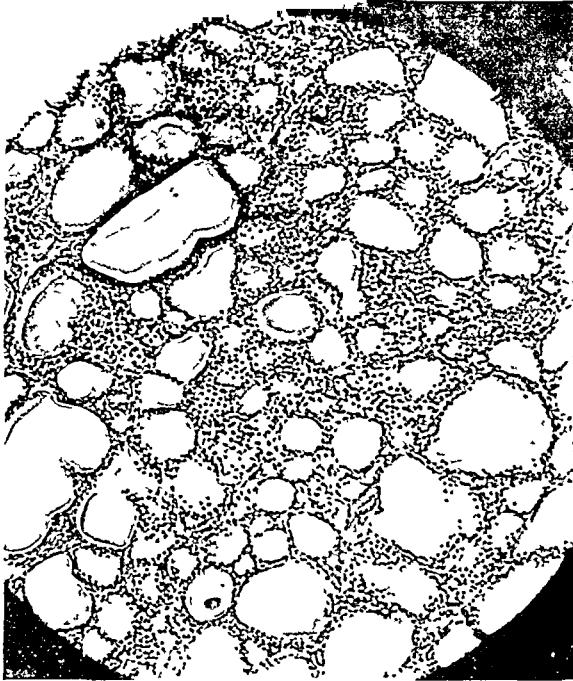


FIG. 1. Low power photomicrograph of normal thyroid gland of dog.



FIG. 2. High power photomicrograph of normal thyroid gland of dog.



FIG. 3. High power photomicrograph of thyroid gland of dog after twelve weeks of thyroid feeding.

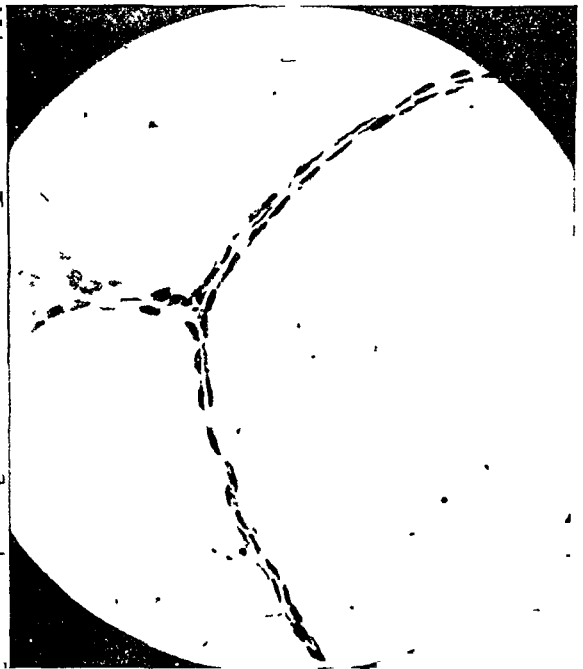


FIG. 4. High power photomicrograph of thyroid gland of dog after feeding desiccated thyroid.

on'y moderately d'stended with colloid. Some of the cells were flattened but many were cuboidal. It would appear as though

the ingestion of desiccated thyroid the sections showed a very definite hyperplasia of the cells. In many areas the

lining membrane had entirely disappeared and the cells were found protruding into the acini. Some acini were completely

It is interesting to note the degree of regeneration which occurs when the animal is given another rest period without iodine

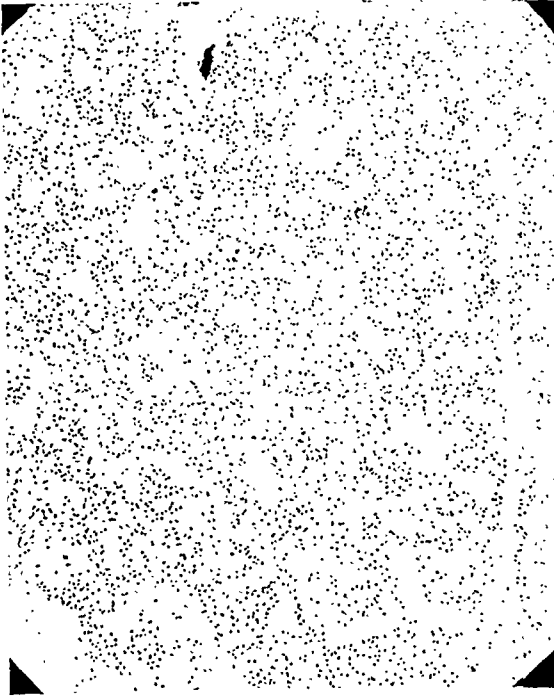


FIG. 5. Low power photomicrograph. Stage of exhaustion.

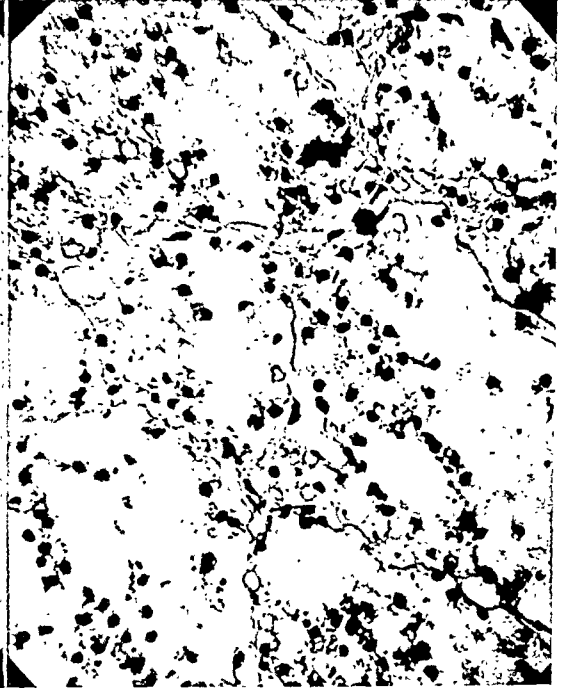


FIG. 6. High power photomicrograph. Stage of exhaustion.

filled with cells. (Fig. 4.)

The final sections are very interesting. Many of the sections taken either from animals that had received iodine alone for both feeding periods or for one feeding period followed by a prolonged rest show varying degrees of thyroid exhaustion. The cells lie free in the acini. The lining membrane has vanished and often all that is seen is a heterogeneous mass of disintegrated cells (Fig. 5). Under higher magnification the cells are granular and the cytoplasm is vacuolated. (Fig. 6). We have interpreted this as a stage of exhaustion of the gland. It is interesting to compare and note the similarity of this photomicrograph to the microscopic picture occasionally seen in the hyperplastic toxic goiter of the human who has been operated on after prolonged iodine administration. The similarity is striking. Marine has seen a similar histological picture in sections removed during the stage of exhaustion atrophy following marked hyperplasia in the human to whom no iodine had been given.



FIG. 7. Low power photomicrograph. Regeneration of dog during period of rest following exhaustion.

(Fig. 7). There is an attempt to reform the normal architecture. Areas of atrophy

adjoin fairly well developed acini containing colloid, while in other areas the cells are just beginning to take on an acinar arrangement. In still other areas the cells are seen to have no definite arrangement. This apparently represents a partial recovery from the stage of exhaustion. Marine has seen the same recovery following iodine administration to cretins whose glands show exhaustion atrophy.

The element of compensatory hypertrophy must be considered in this tendency toward recovery. It has been established that removal of an excessive amount of thyroid tissue will produce a compensatory hypertrophy. We do not believe however that enough thyroid tissue was removed or destroyed from the repeated biopsies to produce such an hypertrophy. It is generally agreed that at least three-fourths of the gland must be destroyed before compensatory hypertrophy will result. Marine³ has shown that ingestion of thyroid substance will prevent this hypertrophy. Concerning the affect of the administration of iodine of regeneration there is a considerable difference of opinion from the conclusions drawn by various observers. Marine has shown that regeneration will not occur in the dog when iodine is administered providing not more than three-fourths of the gland is removed. Loeb⁴ on the other hand has shown that administration of iodine to the guinea pig after partial thyroidectomy not only did not prevent regeneration but actually hastened it. Else has lately confirmed Marine's observations but his animals were from a goiterous region and may therefore be subject to error.

INTERPRETATION OF RESULTS

There is a constant typical effect on the histological picture of the thyroid gland of the dog when iodine is ingested in large quantities. It produces an increase in the amount of colloid with a striking compression of the cells. Marine has demonstrated that nearly 5 per cent of a single dose of

iodine given to a dog is stored in the thyroid gland within a period of two hours from the time it is ingested. He has further demonstrated that the amount of colloid varies proportionately with the iodine content of the gland.

The sections showing disintegration of the cells was interpreted as a stage of exhaustion resulting from prolonged colloid production or from prolonged stimulation of the cells with iodine.

In the animals given desiccated thyroid substance the moderate compression of the cells is due to an increase in the amount of colloid. This may be due to a response of the cells to thyroid ingestion or possibly to the iodine content of the desiccated thyroid.

The hyperplasia which occurred after thyroid feeding would appear to be due to a specific reaction of the gland. The possibility of compensatory hypertrophy must of course be considered.

CONCLUSIONS

1. We have confirmed the observations of Marine and ourselves that ingestion of iodine increases the amount of colloid in the thyroid gland of the dog.

2. Colloid retention compresses the cells lining the acini.

3. A stage of exhaustion may occur in the thyroid of the normal dog after prolonged iodine administration. This may be followed by a partial recovery during a rest period.

4. In this study we were unable to confirm our previous findings that the effect of the ingestion of desiccated thyroid substance produces a similar picture to that found after iodine feeding.

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UROLOGICAL PROBLEMS

OF INTEREST TO THE GENERAL PRACTITIONER^{*}

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BALTIMORE

IN this paper I shall discuss urological problems of interest to the general practitioner, and in so doing it will be necessary to go over much ground that is not new. I will start with the prostate.

Figure 1 is an anatomic plate showing the relation of the prostate to surrounding structures. As seen here the prostate is a very closely encapsulated organ. It is completely surrounded by fascia anteriorly and posteriorly, the posterior being in two layers, the anterior and posterior layers of Denonvilliers' fascia. These layers in fetal life are said to be separated by peritoneum. There are no lymphatics found passing from one to the other, and this is probably the reason why only late in the disease do carcinoma and also tuberculosis penetrate the posterior layer and invade the rectum. The fact that these diseases remain so long confined within the encircling fascia of the prostate makes it possible to obtain a relatively large proportion of radical cures in carcinoma. To reach the prostate through the perineum it is necessary to go through the posterior layer of Denonvilliers' fascia near the apex. Unless this is done there is danger of injuring the rectum.

PROSTATIC HYPERTROPHY

Figure 2 shows a cross section of very early prostatic hypertrophy. One sees here small spheroids which are formed adjacent to the urethra on each side. With time other spheroids form, coalesce, and as they grow larger, become encapsulated, the normal tissue being pushed away from the urethra which is flattened between the hypertrophied lateral lobes (Fig. 3). This adenomatous hypertrophy develops in the lateral and median lobes, but not in the posterior lobe which is represented by a

flat layer of prostatic tissue immediately beneath the posterior capsule, and separated from the lateral lobes by a thin fibrous layer. It is interesting to know that carcinoma almost always develops in this posterior subcapsular prostatic tissue, and in about 50 per cent of the cases in which carcinoma is present, there is a benign hypertrophy of the lateral lobes separate and distinct from the carcinoma of the posterior part of the prostate.

The problem presented the general practitioner in cases of prostatic obstruction is first to make a diagnosis between the benign and malignant disease. If induration of extreme degree, almost stony hard or cartilaginous is present, although it involves only a small area of the prostate in a patient past forty-five years of age, it should usually be viewed with suspicion, and if it is impossible to make a positive diagnosis, an early perineal operation to expose the prostate, inspect and palpate it and if necessary, to excise a portion for microscopic diagnosis should be advised.

Another problem which the general practitioner has to meet is the question of whether there is residual urine present, and whether catheterization should be carried out. Owing to the fact that a simple catheterization is often followed by complete retention of urine, one should usually not catheterize a case of prostatic obstruction unless he is in a position to treat the patient continuously afterwards. It is often possible by palpation and percussion of the lower abdomen to determine whether the bladder is distended. If it is palpable well above the symphysis, one may surmise that 400 or 500 c.c. of residual urine are present, and if it reaches the umbilicus there are generally 1200 c.c.

^{*} Read before the Medical Society of the County of Kings, Brooklyn, N. Y., October 28, 1927.

in the bladder. Large distentions are also palpable per rectum and are usually associated by back pressure effects which

catheter is inserted (in these patients with evidence of large residual urine) the rubber tube running to the lower end of



FIG. 1. Fascia surrounding prostate, seminal vesicles and ampullae of the vasa deferentia.

produce the symptoms of uremia and are to be detected by chemical examination of the blood. In passing a catheter in cases of prostatic hypertrophy it must be remembered that the urethra is flattened from side to side by the lateral enlargements and then pushed upward by the intravesical median lobe which may produce such a pronounced bend in the urethra that a plain soft rubber catheter or even an ordinary silver catheter will fail to pass. The use of a Coudé catheter, either of rubber or gum-coated linen, will usually facilitate catheterization in these cases. When a catheter is passed in patients with a large amount of residual urine, e.g. over 500 c.c., complete evacuation of the bladder should not be allowed, lest hemorrhage or complete suppression of urine supervene. The mere emptying of the bladder in a case where 1000 c.c. are present is often followed by severe shock with a very great drop in blood pressure and not infrequently sharp bleeding. Uremia is apt to follow and is due both to the drop in blood pressure and to the sudden change in intrarenal pressure which upsets the secretory balance.

In order to avoid such occurrences we employ gradual decompression by means of special apparatus. As soon as the

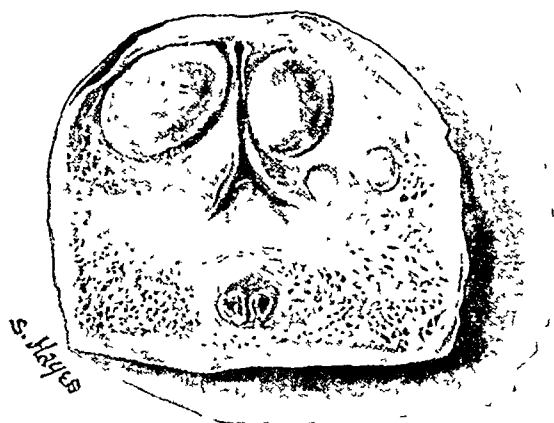


FIG. 2. Cross section of an early hypertrophy of the prostate. In lower portion are visible ejaculatory ducts and dilated orifices of some gland acini.

the instrument is immediately connected up with a catheter in the bladder and the intravesical pressure determined by noting the point at which the urine ceases to flow over the overflow tube. Turning the wheel on the ratchet the overflow tube is

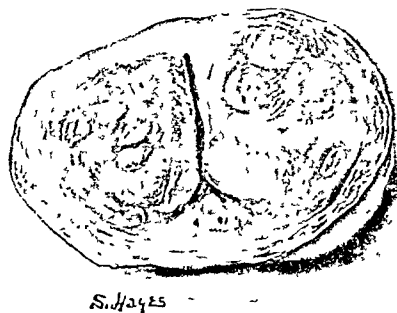


FIG. 3. Cross section of more advanced prostatic hypertrophy.

placed at a point 4 or 5 cm. below the intravesical pressure and the urine allowed to escape into a bottle. Every few hours the height of the overflow tube may be reduced so that within two or three days it is usually possible, even in cases of great overdistention, to lower the outflow tube to zero and then remove the apparatus. Shaw has added a reservoir to my apparatus so that an antiseptic solution may be allowed to flow in and out of the bladder and combat infection.

During this critical period in which an overdistended bladder is being decompressed, frequent blood chemistry tests should be carried out to note the changes in the urea. Uremia is combatted by appropriate cardiac stimulants; large amounts of water, either by mouth, subcutaneously or by rectum; the use of rectal or intravenous glucose and other supportive measures. As soon as the apparatus is reduced to zero a phthalein test may be carried out to check with the blood chemistry. Continuous catheter drainage is kept up until the phthalein test shows sufficient improvement in the renal function and examination of the cardiovascular system indicates that the patient may be successfully operated under caudal or local anesthesia.

The problem which confronts the general practitioner is whether, first of all, a case of prostatic hypertrophy may be allowed to go longer without catheterization or operation. During this period he should be seen from time to time and careful examinations made to be sure that he is not developing considerable residual urine with back pressure effects upon the kidney and cardiovascular system. This can usually be determined without catheterization. If infection of the urinary tract supervenes it is usually not safe to delay more vigorous treatment, either by catheterization or by operation, lest pyonephrosis develop. When urination is frequent, difficulty and obstruction evidently increasing, the general practitioner must decide between a catheter life and operation. Extensive statistics show conclusively that the catheter life is dangerous and is attended ultimately by a mortality of 10 per cent or more. The consensus of opinion is that operative treatment should be undertaken if the patient can be got into sufficiently good condition as regards the kidneys, heart and arteries. It is amazing what improvement can be brought about by continuous or intermittent catheterization, in addition to general supportive measures, as just described. Even where

the kidneys are greatly impaired and definite uremia associated with pronounced cardiovascular disturbance is present, although it may take weeks, such cases can usually be prepared satisfactorily to undergo an operation under caudal anesthesia through the perineum. It is my deliberate opinion, after years of experience, that the perineal route is not only the safest, but the most anatomical, in that it is possible to reach the prostate directly without going twice through the bladder. The dependent drainage and the lessened chance for infection also make the mortality rate distinctly lower than by the suprapubic route. In cases of hypertrophy one does not actually carry out prostatectomy. The operation, either perineal or suprapubic, is not a removal of the prostate, but enucleation of the adenomatous enlargement, an intraprostatic adenectomy, leaving behind the normal prostate, which has been pushed aside by the intraurethral growth of lateral and median hypertrophied masses.

Caudal anesthesia is certainly far preferable to general anesthesia, and also to spinal anesthesia and local block anesthesia. The simple introduction of only 20 c.c. of a 3 per cent solution of novocaine is sufficient, in almost all cases, to give complete and satisfactory anesthesia to carry out not only enucleating prostatectomy through the perineum, but also a radical operation for cancer of the prostate or for tuberculosis of the prostate and seminal vesicles.

Most of the difficulties which have been encountered in perineal prostatectomy have come from failure to do an anatomic operation, to recognize the normal structures, and reach the prostate without anatomical and physiological injury. Through an inverted U incision one first exposes the space on each side of the central tendon back of the transversus perinei muscles. The central tendon is then divided, and the rectourethralis muscle which lies in the median line and runs from the rectum to the membranous

urethra (Fig. 4) is then exposed. It is made taut by picking it up with forceps (Fig. 4a) and divided close to the mem-

incision, which preserves the floor of the urethra, or an inverted v incision in which the lateral and median lobes are removed



FIG. 4. Exposure of rectourethral muscle (center) a, Its division. b, Urethrotomy. c, Elevation of cut edges of urethra.

branous urethra, after which the membranous urethra back of the external sphincter is easily exposed. The rectum, freed from its attachments by the division of the rectourethralis, is easily pushed back, exposing the apex of the prostate, while the external sphincter, bulb and Cowper's glands are drawn forward by means of a grooved retractor (Fig. 4b). In this figure the incision of the urethra upon a sound is shown. The edges of the mucous membrane are then elevated with the point of the scalpel and caught with Allis clamps (Fig. 4c) previous to the introduction of a prostatic tractor, by means of which the prostate is drawn downward and the posterior surface freed from its attachments to the rectum (Fig. 5). With the prostate thus well exposed, one is prepared to examine areas suspected of malignancy and if unable to make a diagnosis, to excise them for immediate microscopic study. When the case is evidently benign one may carry out enucleation of the hypertrophied masses through either a bilateral capsular

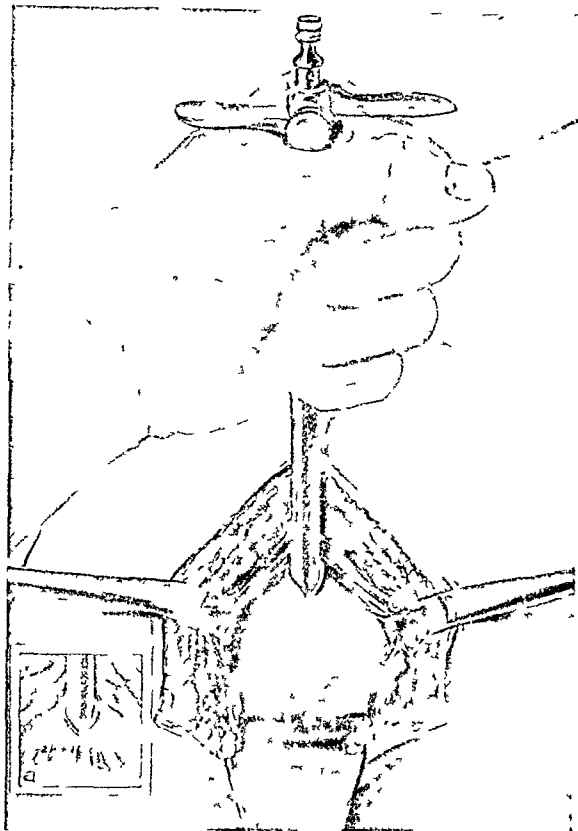


FIG. 5. Tractor inserted and opened; posterior surface of prostate exposed, showing anterior layer of fascia of Dénonvilliers. a, before rectum is pushed back.

in one mass (Fig. 6) I will not go into the details of the technique. In some cases with hypertrophy present in the anterior commissure, it is advisable to remove this with the lateral masses. By means of this operation one is able to see and avoid the verumontanum and ejaculatory ducts, a very important factor not only in avoiding epididymitis, but also in preserving the sexual puissance of the patient, and it is surprising to know how many, even very old patients, lay stress on this asset.

With the removal of the lateral and median hypertrophied masses, one may adopt various forms of closure. The simplest is to put in a rubber tube through the perineum and pack the lateral cavities. A recent modification has been the use of

the Davis hydrostatic drainage bag which stops hemorrhage by traction. More recently I have been introducing a catheter

anterior fascia, which closely hugs the prostate, is stripped upward carrying with it the vessels and nerves in the pubopro-



FIG. 6. Middle lobe attached to two lateral lobes, already freed, is being separated from sphincter and vesical mucosa and enucleated from behind forward

into the urethra and closing the prostatic wound completely by means of sutures which are placed so as to include the vesical orifice and stop the hemorrhage as well as obliterate the lateral and median posterior cavities and approximate the capsular wounds. In about 100 cases we have been able to get *per primam* healing and no leakage through the perineum in about 60 per cent of the cases.

CARCINOMA

Where carcinoma is recognized early enough for radical operation, either by simple examination or inspection and resection of a portion after perineal exposure, the radical operation offers excellent results in a large percentage of the cases which are appropriate to this procedure. As seen in Fig. 7 the side of the prostate is exposed by pushing aside the fascia which covers the side and anterior surface of the prostate. The membranous urethra is then divided (Fig. 7a) and the

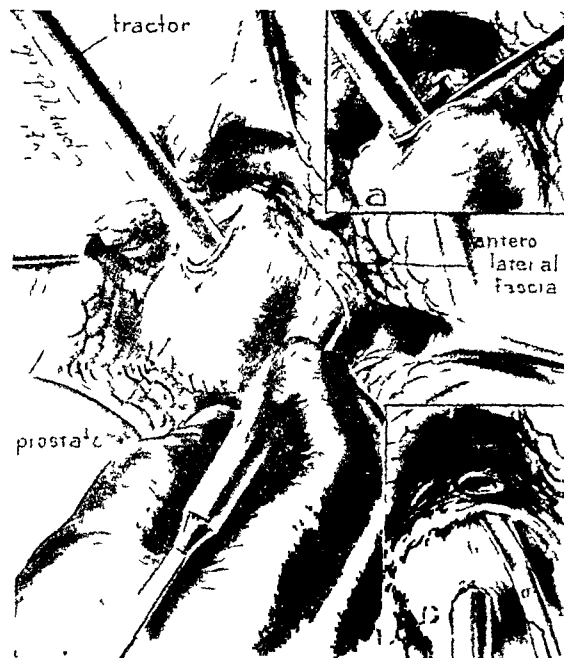


FIG. 7. Radical operation for prostatic cancer. Anterior fascia is stripped from prostate (center). Membranous urethra divided (a). Anterior surface of prostate exposed to vesicoprostatic juncture (b)

static region. This makes it possible to expose the anterior surface of the bladder at the vesicoprostatic juncture (Fig. 7b). Figure 8 shows in detail the successive steps in exposing, ligating and excising the seminal vesicles and vasa, along with the prostate. No difficulty is experienced in getting satisfactory anastomosis and complete closure of the perineal wound.

In the 27 cases in which we have carried out this radical operation we have not been troubled with strictures or fistulae, and a careful study of ultimate results shows that over 70 per cent of the patients who have left the hospital have remained well for periods of from five to thirteen years. By the most recent technique, in which the vessels and nerves of the pre-vesical space are avoided, excellent urinary control and normal urination have been obtained in most of these cases, even though the entire prostate, neck of bladder and internal sphincter have been removed.

If the medical profession could be interested in the early diagnosis of carcinoma of the prostate, far more cases

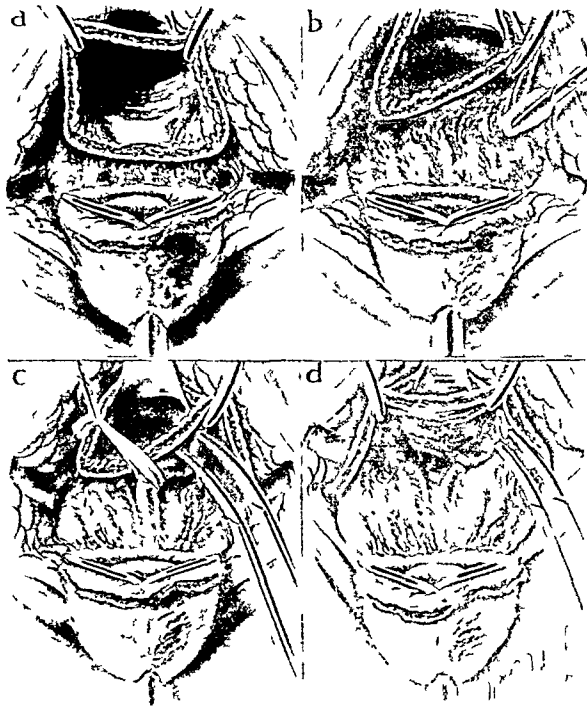


FIG. 8. Trigone divided (a), Bladder stripped up from vesicles and vasa (b), which are ligated (c) and pedicles divided (d).

suitable for radical operation would come to light. It should be remembered that in men past forty-five years of age, when one examines by rectum and finds an area of very marked induration, even though it involves only a small part of the prostate, carcinoma should be thought of. If this induration is of almost stony hardness, although it seems smooth and encapsulated, carcinoma is probably present. The diagnosis will be made positive by exposure through the perineum and, in view of the excellent results obtained by radical operation, this should always be advised. Where the carcinoma has progressed too far for radical operation radium may sometimes be employed through the urethra and rectum with splendid effect. This is particularly true where repeated hemorrhages have occurred. Bleeding is often immediately stopped by a single application of radium. For this purpose

we employ an applicator as shown in Figure 9, in which two tubes of 100 mg. radium element each are carried, tandem,

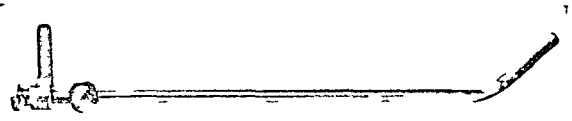


FIG. 9. Radium instrument used for the treatment of cancer of prostate. Instrument carries an observation cystoscope for use in bladder tumors

in the beak. This instrument is introduced into the urethra and held in the proper position by means of a clamp attached to the frame of the table. The same instrument may be applied against the posterior surface of the prostate through the rectum, under the guidance of a gloved finger, and held in the appointed place by means of a clamp attached to the table. Introducing the same instrument into the bladder, and turning it to the right or left and elevating the handle, it is possible to bring the beak against the trigone and to radiate the carcinoma in the region of the seminal vesicles through the bladder. By intelligent and careful applications through the urethra, bladder and trigone, it is possible to give a considerable amount of radium without producing ulceration of the mucous membrane. The important point is to see that it is never applied twice to the same region. I have carried out radium treatment in about 400 cases with results which often have been extremely satisfactory in stopping hemorrhage and removing obstruction. I am not able to say, however, that any case has been permanently cured, and I am more than ever convinced that the radical operation should be carried out when feasible. Owing to the fact that the radium treatment is protracted, it is often more satisfactory to carry out the conservative perineal prostatectomy described, but in cases with bleeding, pain and moderate obstruction with marked debility, radium is a godsend and generally very satisfactory.

CONTRACTURE OF THE VESICAL ORIFICE OR MEDIAN PROSTATIC BARS

Figure 10 shows the condition usually

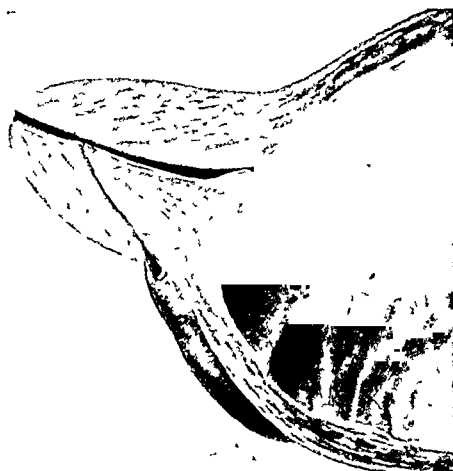


FIG. 10. Longitudinal section showing typical median bar elevated above trigone without enlargement of prostate.

met in these cases. As seen here, the posterior prostatic margin is elevated by a transverse bar and this is usually associated with circular contracture of the vesical neck. This condition should be

lobes. Where the contracture occurs alone, it may be satisfactorily dealt with by an operation which I presented in 1909 and



FIG. 12. Author's punch in operation. Outer tube has been withdrawn far enough to entrap median bar in fenestra, as indicated by a checking in flow of fluid. Hands of operator are shown ready to push inner tube home.

which has since been known as the punch operation. The contractures of the vesical orifice or the median bars may be congenital or develop in childhood and are most common between the ages of thirty and forty-five, although they do occur in the age of prostatic hypertrophy. The

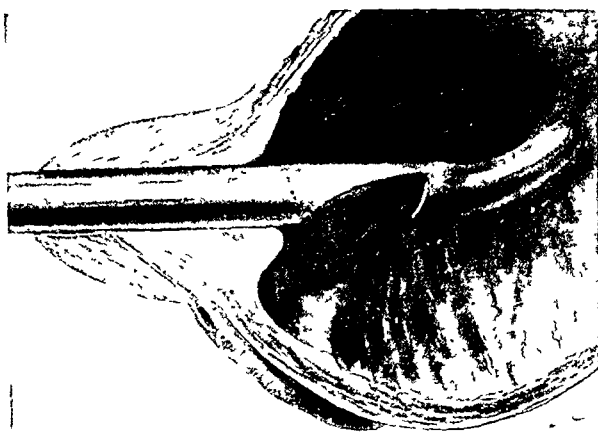


FIG. 11. Punch instrument introduced well into bladder and inner tube drawn upward, thus opening fenestra through which urine begins to escape. Median bar is seen depressed by shaft of instrument.

sharply differentiated from cases of prostatic hypertrophy. The two may occur together, but in such cases the proper treatments should be that appropriate for the removal of hypertrophied prostatic



FIG. 13. Punch operation. Cutting inner tube pushed in, excising bar caught in fenestra.

symptomatology may be identical with that of hypertrophy, although hematuria usually occurs less often and sexual symptoms and other evidences of chronic prostatitis and seminal vesiculitis are more frequently seen. The diagnosis is

made by rectal examination, cystoscopy, palpation with finger in rectum and cystoscope in the urethra, showing an indurated collar around the vesical orifice, the presence of residual urine, trabeculation, back pressure effects, etc. The punch operation makes it possible to remove a median bar or a generally contracted orifice under local procaine anesthesia. The instrument is introduced into the bladder and the inner cutting tube withdrawn, thus opening the fenestra (Fig. 11) and allowing the fluid to escape. Quickly pulling the instrument outward, it is arrested as the median bar drops into the fenestra (Fig. 12). Pushing the inner cutting tube home, the bar within the fenestra is completely excised (Fig. 13). Similar cuts may be made on each side and even anteriorly, in case there is an anterior valvular obstruction present (a condition that occurs not infrequently). Hemorrhage, which may be fairly abundant, is stopped by introducing a spiral cautery through the outer tube and immediately cauterizing the cut area. A catheter may or may not be introduced for drainage, depending on the continuation of the bleeding. I have also carried out this operation by means of an electrocautery punch which has been modified by Caulk, who has produced a simpler instrument without my irrigating sheath which he has employed in a great number of cases with excellent results. Recently various other modifications of my punch instrument have been brought out in which diathermy and the radio knife have been introduced.

The punch operation has furnished an excellent method for the radical cure of these fairly common cases of obstruction at the vesical orifice in younger men not the subjects of prostatic hypertrophy. The same operation has been used in many cases of carcinoma of the prostate where the obstruction was in the form of a collar at the vesical orifice. The results obtained are often quite good, but I believe the conservative removal through the perineum is better.

CONGENITAL VALVES OF THE PRO- STATIC URETHRA

This is a subject of great interest which has received very little attention. The first case detected clinically and radically cured by operation was one which came to me in 1912. A child with dribbling of urine presented a greatly distended bladder and on attempting to pass a catheter, obstruction was met with in the prostatic portion. As no instrument could be introduced into the bladder, I carried out suprapubic operation and found the vesical orifice widely dilated and a membranous diaphragm obstructing the sound which had been introduced through the penis. Removal of this diaphragm resulted in immediate cure. Since then I have operated on about a dozen cases, some by the suprapubic method and others by means of a "baby punch" quite similar in design to the larger instrument which I employ for median prostatic bars, as already described. The diagnosis of this condition is relatively easy. These patients present, from birth, evidence of marked obstruction to urination, a distended bladder and often great distention of the ureters and kidney pelves associated with pronounced uremia and a marasmic condition. Many cases have died shortly after birth, and some that have come to us have been beyond relief. When children with incontinence of urine are seen, one should think of the possibility of these congenital valves, especially if the health of the child is gravely impaired and a distended bladder is made out. In such cases it is not safe to do a suprapubic operation at once. Gradual deflation and decompression of the bladder are essential to avoid death from uremia. We found a fine ureteral catheter very satisfactory for such gradual decompression. It is usually possible to introduce such an instrument through the slit which is usually present between the two halves of the diaphragm which runs from the verumontanum to the lateral walls of the urethra. Blood chemistry and phthalein tests will give an index

as to preliminary treatment which should be given just as in cases of prostatic hypertrophy when the patient is suffi-

in accepting the fact that tuberculosis of the epididymis is most commonly secondary to or accompanied by tubercu-

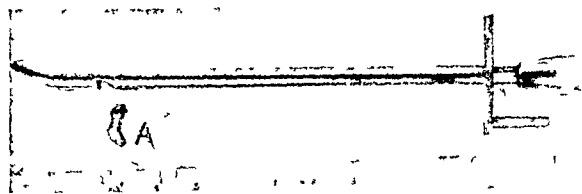


FIG. 14. Specially designed punch for removal of congenital valves in young children.

ciently improved for operation. In recent years we have found the operation with the baby punch entirely satisfactory. It can be done with local novocaine or without anesthesia if the child is held still. It takes only a minute to introduce the instrument, withdraw the inner cutting tube, draw out the outer sheath, entrap the valve in the fenestra and quickly excise it by pushing home the inner cutting tube. Figure 14 shows the baby punch with a diaphragm which had been excised in one of these cases. Figure 15 shows a cystogram in which the great dilatation of the kidney pelvis, ureters and bladder as well as the upper half of the prostatic urethra above the valve is evident.

When we made our first report there were no cases in the literature except those which had been discovered post mortem. We were first able to report 12 complete cures in 18 patients. Six were too far gone for operation on entrance to the hospital. All operated upon got well. Since then we have had perhaps a dozen more cases. Recently Hinman has reported successful cases upon which he has operated. The subject is one which has gained surprisingly little interest and I wish to bring it especially to the attention of pediatricians and those in general practice who see many cases of this disease in childhood.

TUBERCULOSIS OF THE PROSTATE AND VESICLES

The medical profession has been slow



FIG. 15. Pyelo-uretero-cystogram in case of congenital valves of urethra, showing dilatation of entire upper urinary tract.

losis of the seminal vesicles and prostate. Overwhelming statistics have been piled up in recent years which show definitely in my opinion that it is irrational to expect radical cures by epididymectomy in tuberculosis of the epididymis. For many years we have advocated a radical operation, in which, in addition to the epididymis and vas deferens, the seminal vesicles and lateral lobes of the prostate are removed through the perineum. We have recently shown that this can be carried out under caudal anesthesia, thus making the procedure safer on account of the not infrequent presence of pulmonary tuberculosis. By means of a special long tractor it is possible to expose the prostate and seminal vesicles through an incision similar to that which I employ in prostatic hypertrophy (Fig. 16). Incising the fascia which covers the prostate and seminal vesicles, as here depicted, the lateral lobes, vesicles and vasa are brought to view. The seminal vesicles and vasa are then separated

from their attachments and drawn downward and removed in one piece with the lateral lobes of the prostate (Fig. 17), care

with the cautery (Fig. 18). In order to remove the vas completely, traction is made upon the vas as it emerges from the

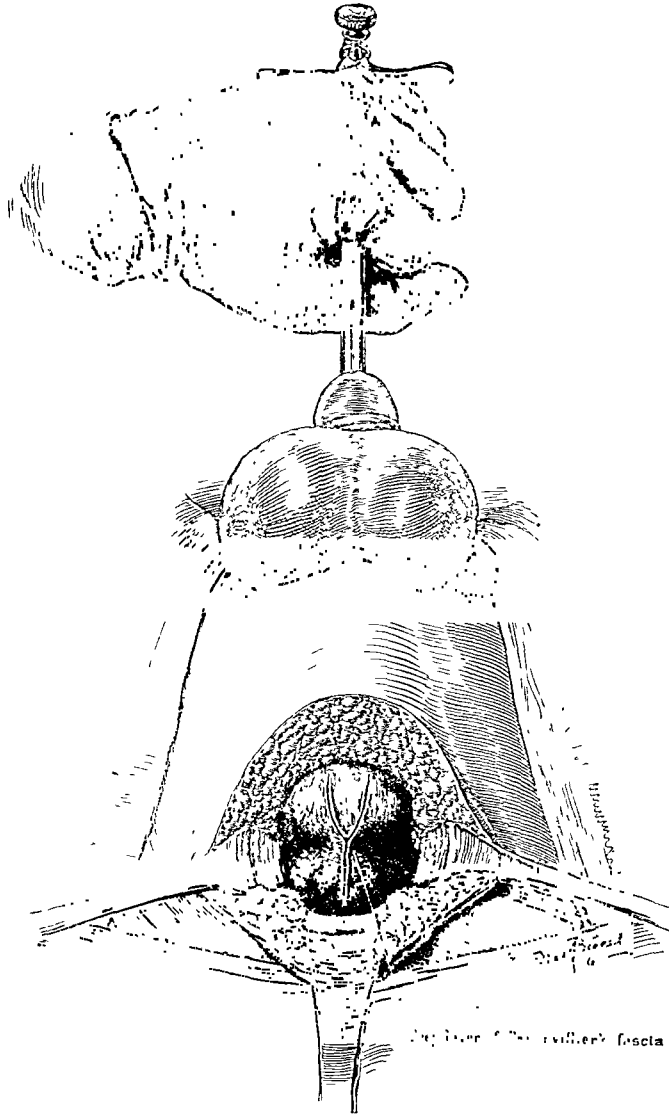


FIG. 16. Levators and rectum drawn away, exposing fascia of Dénonvilliers. Y-shaped incision through fascia.

being taken to avoid opening the urethra or bladder. This is not a difficult procedure and has now been carried out by me in about 25 cases most satisfactorily. After removal of the vesicles and lateral prostatic lobes an incision is made in the scrotum to remove one or both epididymes, as may be necessary. As the testicle is usually not involved this is left behind after carefully separating the vas from the veins, and cutting off the globus major from the upper end of the testicle

external ring and intermittently upon a clamp which has been placed upon the cut end of the vas in the perineal wound (Fig. 19). In this way it is freed and then drawn outward through the groin after removing the perineal clamp. Figure 20 shows a case in which the left epididymis and vas, the right testicle, epididymis and vas have been removed with the seminal vesicles and lateral lobes of the prostate. This was accomplished without opening the urinary tract and with excel-

lent result, which has now been maintained for ten years. In 25 per cent of the cases tuberculosis of one kidney has been

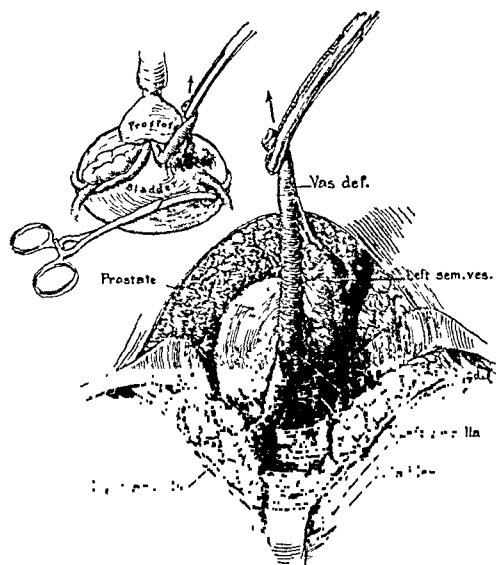


FIG. 17. Left seminal vesicle freed, vas divided between clamps.

present and nephrectomy carried out. Several of these patients in whom the kidney, both epididymes, both seminal vesicles, both vasa and both lateral lobes of the prostate have been removed, have been well now for more than five years. Careful study of our cases in which the palliative procedure of epididymectomy or castration alone was carried out has proved, to my mind conclusively, that where pronounced involvement of the seminal vesicles and prostate is present, only by a radical procedure as already described can curative results be obtained. When very slight involvement of one seminal vesicle is present it is quite possible to inject carbolic acid through the vas after removal of the epididymis and obtain satisfactory results in some cases. This is the procedure which we employ when we think the radical operation is not necessarily indicated.

MERCUROCHROME IN URINARY AND GENERAL INFECTIONS

There is no other branch of medicine in which the fight against infection so engrosses the physician as in the genito-

urinary tract. In addition to the widespread venereal infections, we encounter inflammations of the kidney, ureter, blad-

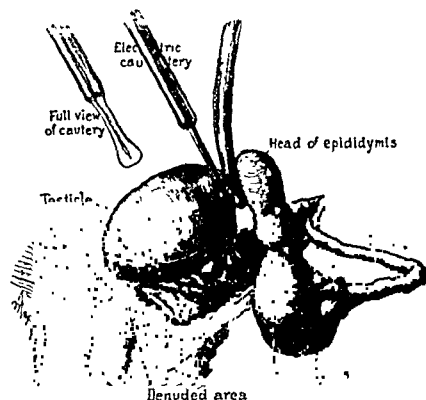


FIG. 18. Excision of epididymis completed with electrocautery.

der, prostate, vesicles, epididymes and testicles as a result of infections with many different types of organisms. I have been greatly interested in this subject for many years and with the organization of the Brady Urological Institute, some thirteen years ago, installed chemical and bacteriological laboratories for research on these important subjects. During the War I visited the laboratories and hospitals in England and France where special research on antiseptics was being conducted. On returning from the War I renewed our attack in the laboratory and clinic. Working with us, Drs. White, Swartz, Davis, Colston, Hill, Scott, and others have conducted an extensive series of researches. Dr. White, chemist to the Brady Urological Institute, has prepared a long series of drugs which have been tested in the laboratories for their bactericidal strength and their efficiency in the presence of blood serum and urine. Commencing first with an effort to make an antiseptic compound with phenolsulphonphthalein, with an idea of getting germicidal elimination through the urinary tract, the research has spread into a study of all available dyestuffs and their compounds, and from this several drugs of definite value have been produced. I will only mention No. 220 of our series

to which we have given the name mercurochrome, and concerning which we have published numerous papers, so I need not

140 colonies per cubic centimeter. When the condition seemed hopeless, he was given 34 c.c. of a 1 per cent solution of

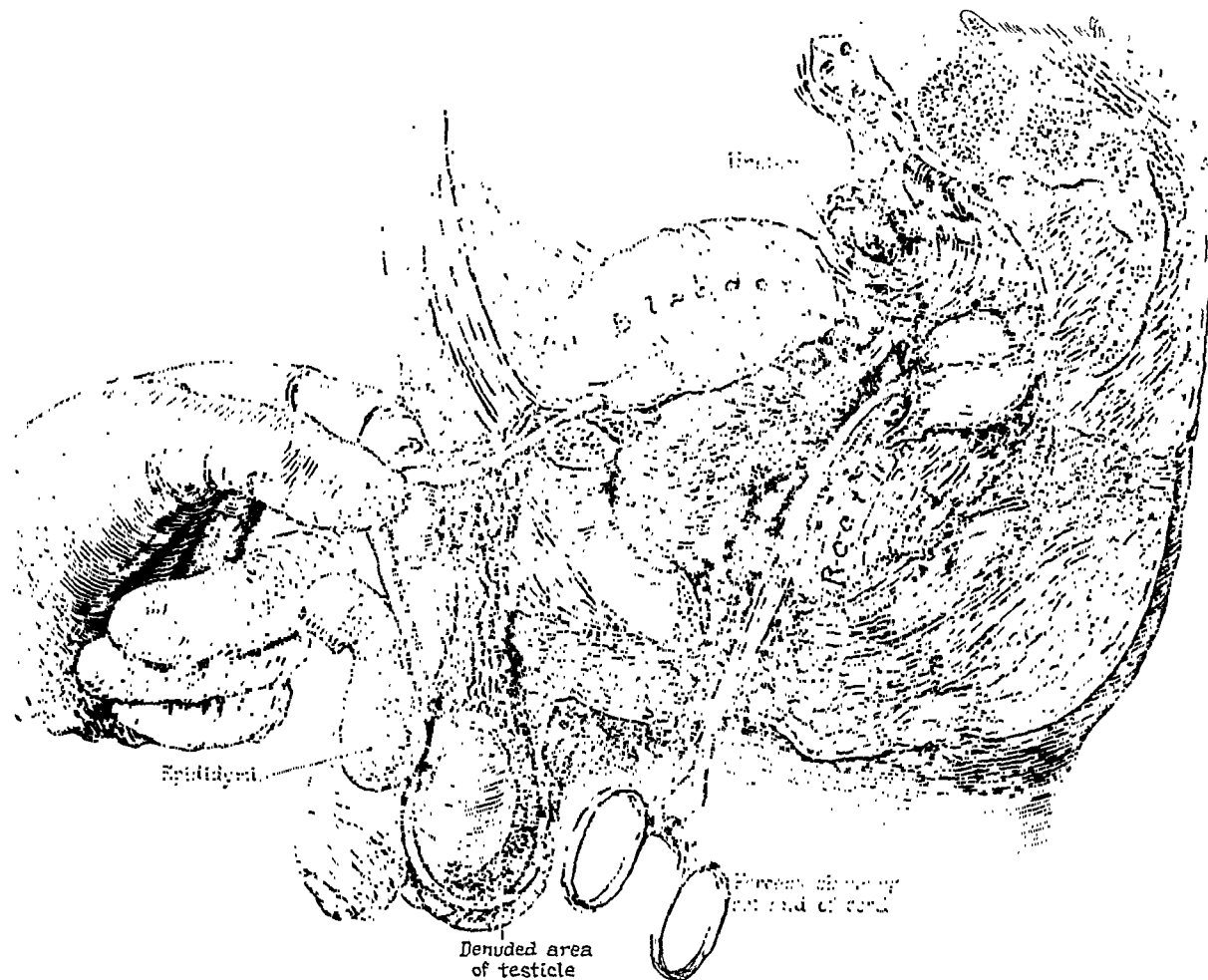


FIG. 19. Method of traction (alternating between operator in groin and assistant pulling on clamp in perineal wound on vas) by which vas is freed and then drawn out through inguinal canal.

go into detail here. Being a mercurial of high germicidal potency which is active also in the presence of serum and urine, it has been employed very widely in the various specialties of medicine for the local treatment of inflammatory conditions. For the last five years we have used it more and more frequently as an intravenous germicide. The first case in which its value was positively demonstrated was that of a man who came in with bilateral pyonephrosis due to the *Bacillus coli communis*. Following ureter catheterization he developed septicemia and rapidly became worse, so that his life was despaired of. A blood culture gave

mercurochrome (5 mg. per kilogram body weight). This was followed by chill, rise of temperature to 105°F. and a rapid drop, with startling improvement in the patient. The blood was sterile the next morning and in a few days the patient was well. The urine was free from infection. Figure 21 shows the temperature curve in this case.

Another case which showed the value of the drug in local infections was that of a young man who following instrumentation developed a retrovesical infection which rapidly travelled upward and surrounded the kidney as a perinephric abscess, characterized by a large area of induration, marked tenderness and high

temperature. The organism was *B. coli*. Twenty-eight cubic centimeters of a 1 per cent solution injected intravenously, was



FIG. 20. Photograph of specimen showing removal of both vesicles, vasa and lateral lobes of prostate; median portion and urethra preserved. Castration on one side, epididymectomy on other.

followed by a rapid drop in temperature to normal and a prompt disappearance of the inflammatory mass.

Another remarkable case was that of a boy aged four who came into the Sydenham Hospital with scarlet fever associated with marked tonsillitis, pharyngitis, cervical adenitis, facial crysipelas, septicemia and acute nephritis. Blood culture showed 28 colonies of *Streptococcus hemolyticus* per cubic centimeter of blood. Antistreptococic serum gave no benefit and when the case seemed hopeless he was given 15 c.c. of a 1 per cent solution of mercurochrome intravenously (a large dose: 7.5 mg. per kilogram body weight). This was followed by a rapid sterilization of the blood, disappearance of the inflammatory conditions of the mouth with sterilization of the urine and complete recovery in a few days.

The publication of a case of pneumonia which was furnished us by Dr. George Martyn was followed by the introduction of this treatment into the Child's Hospital at Atlanta by Freeman and Hoppe, who have made three reports, showing splendid

results in lobular pneumonia in children with intravenous injections of mercurochrome. In a series of 180 cases the mortality has been reduced from 36.5 per cent to 8.6 per cent and some of the cases reported by them show really amazing cures. The intravenous injection of mercurochrome is generally followed by a sharp drop in the temperature curve, corresponding to that seen in the crisis in pneumonia (Fig. 22), which is a chart of one of their cases.

I have not space here to give in detail the widespread use of intravenous mercurochrome which has resulted from our publications, nor even to summarize the great number of cases in which we have employed it. Suffice it to say that in cases of septicemia of various types, from 60 to 75 per cent of the cases have recovered after intravenous mercurochrome. Its use in the peritoneal cavity when general or localized infection has been found at operation was recommended by two competent surgeons in a long series of cases. Three cases of liver abscess detected by laparotomy, considered hopeless and the abdomen closed, have apparently disappeared completely as a result of intravenous mercurochrome. Several interesting cases of lung abscess have also been reported by Davis, Holden and others. Remarkable visual demonstrations of the efficacy of intravenous medication with mercurochrome have been made in a long series of cases of erysipelas, (Fig. 23), encephalitis, boils, carbuncles, etc. Tally reports 25 cases of carbuncles of the neck treated intravenously with mercurochrome, and without local supportive intervention, with complete recovery in all cases. In other skin infections and some skin diseases not generally recognized as infectious, mercurochrome has been very efficacious. In pyelonephritis due to staphylococcus infection, sterilization of the urinary tract can almost always be accomplished with a few injections of intravenous mercurochrome. It can also be secured in many cases by intravenous novarsenobenzol and

occasionally by the use of gentian violet which we have also promulgated. In *B. coli* and particularly *B. lactis aerogenes*

The experimental work shows that the drug is eliminated within thirty minutes in considerable quantity through the uri-

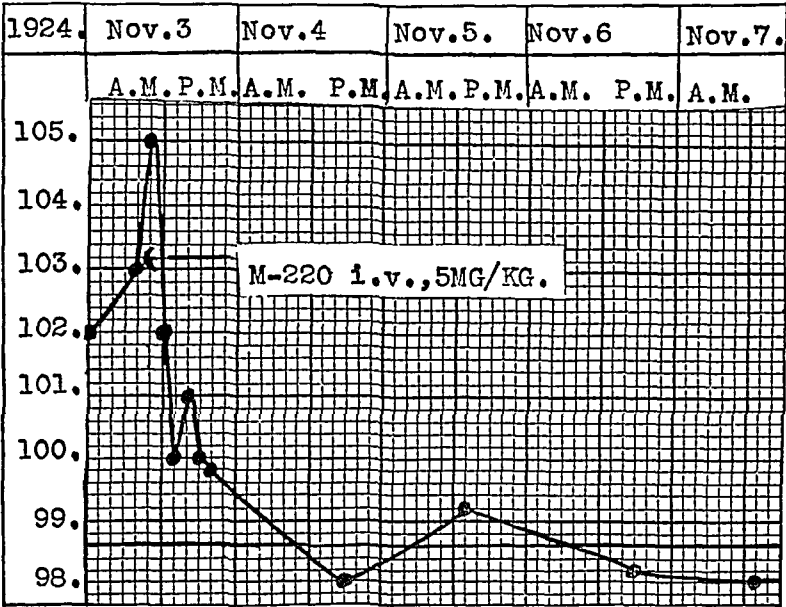


FIG. 21. Man, aged forty-four years. Double pyelonephritis following ureter catheterization. General septicemia. *B. coli*, 140 colonies per c.c. blood. Temperature varied from 103° to 104°F. January 3, 1922, patient irrational. Medical consultants thought case hopeless, and patient would not live more than a few hours. Mercurochrome 1 per cent, 34 c.c. intravenously (5 mg. per kg. body weight). Injection followed by chill, slight rise in temperature and precipitate drop of 6° in about four hours. Blood culture twelve hours after injection sterile. Patient conscious and greatly improved. Ate breakfast. Rapid convalescence. Blood cultures remained sterile. Patient discharged well in seven days.

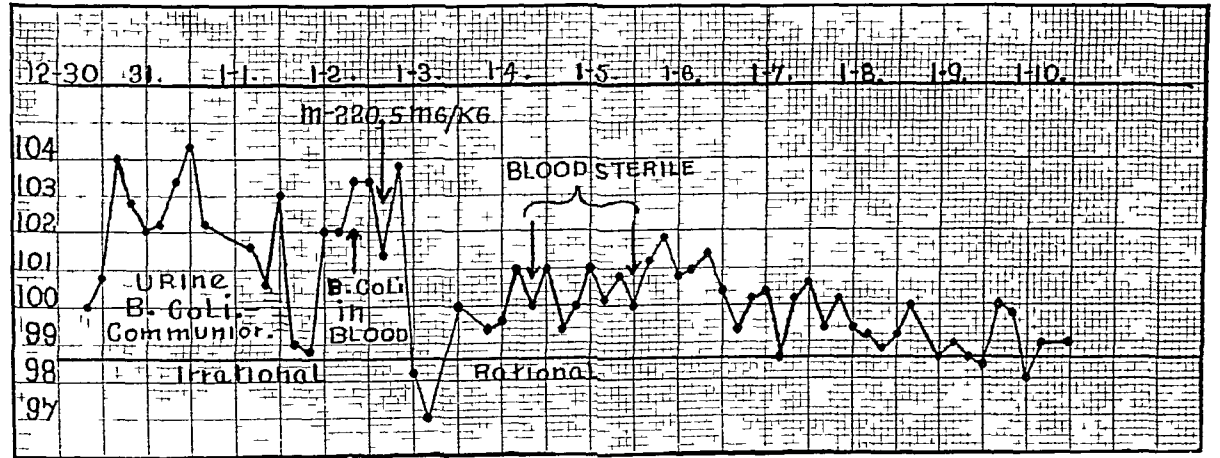


FIG. 22. Girl, aged five and one-half years, developed measles, followed by bronchopneumonia and acute otitis media. Had been ill about six weeks, temperature varying from 102° to 105°F. During fourth week of illness developed severe diarrhea; four to seven stools daily. Thirty-eighth day of illness, condition desperate; five areas of consolidation in lungs, temperature 104.6°F. Marked emaciation; both ears discharging profusely; numerous skin abscesses. Hope for recovery abandoned. Intravenous mercurochrome (5 mg. per kg. body weight) with very little reaction. Temperature fell as if by crisis, and rapid uninterrupted recovery followed.

infections of the urinary tract, the results obtained are less satisfactory, although a number of sterilizations have been obtained.

nary tract, the presence of mercurochrome being easily detectable for more than twenty-four hours. It is eliminated also in a

larger quantity through the biliary tract and experimental and clinical work seems to indicate that it has a distinct value in

to the kidneys from intravenous mercurochrome. A study of autopsies on cases of septicemia that did not receive mercuro-

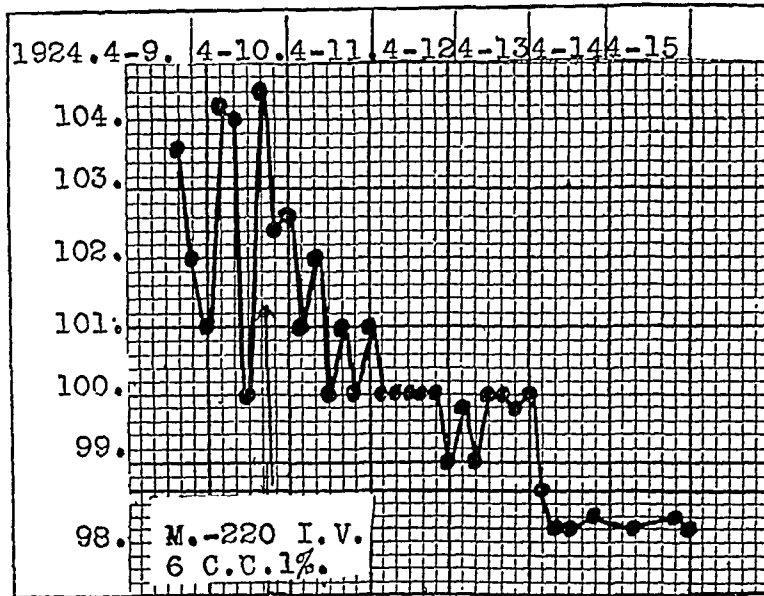


FIG. 23. Erysipelas, mastoiditis, sepsis; condition very grave. Striking benefit from mercurochrome 220, one injection, 25 c.c., 1 per cent.

the treatment of the gall bladder, biliary ducts and even of the liver itself, as indicated above.

GONORRHEA AND ITS COMPLICATIONS

Excellent results have often been obtained by intravenous mercurochrome in arthritis and gonorrheal rheumatism. We found it essential, however, to continue active local treatment of the urethra and bladder in order to obtain a complete removal of the gonococcus. By means of combined local and intravenous therapy we are convinced that gonococcal infections are much more rapidly eradicated than under simple local treatment. Our experience in this has been confirmed by Whitman, Walther, Redewell and others. Very early in our experimental work my associates proved conclusively that the drug could be given even in large doses without fear of injury to the kidneys. A study of many hundred cases, among which Scott reported an analysis of 500, there was found no evidence of injury

chrome shows the same lesions in the kidney which have been found in a few cases which have died regardless of the use of mercurochrome. We feel, therefore, that the attack which has been made upon the use of mercurochrome intravenously as a result of a few isolated cases is absolutely controverted and repudiated. I do not pretend that it is universally successful. There are many cases in which it fails to work, cases similar to others in which good results are obtained, but after an exhaustive study of four years and a collection of many hundreds of cases from my clinic and from sympathetic and cooperative physicians in all parts of the world, I have no hesitation in saying that with care intravenous mercurochrome may be given with impunity, that it brings about a reaction which often terminates abruptly a local or general infection after one or more doses. That it does not produce a germicidal blood serum and thus kill off infection, we now know. Initial doses of 15 c.c. of a 1 per cent solution are

generally satisfactory. The reaction is probably similar to that induced by foreign proteins and is apparently a combined bacterial, cellular, serum and tissue reaction resulting, as a rule, in rise of temperature, followed by a sudden fall. Our inability to explain exactly what happens is no more surprising than the inability of internists and serologists to explain what happens constantly in pneumonia where patients rapidly get well by crisis.

In conclusion I may say without exag-

geration that intravenous antiseptic therapy has been demonstrated to be a valuable therapeutic agent, that various drugs may be employed, that often surprisingly rapid cures or improvements of widespread local and general infections occur, that many unexplicable failures are encountered, but taken as a whole, great progress has been made and much can be accomplished by a careful, systematic, sympathetic use of intravenous therapy, of which mercurochrome is, up to the present, apparently the most valuable example.



PATHOLOGICAL CONSEQUENCES OF THE CONGENITALLY PTOSED RIGHT COLON: SURGICAL TREATMENT IN SELECTED CASES*

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THE thesis of this paper is that the ptosed colon is congenital, and that in most individuals it causes no noticeable dysfunction; but that in a small percentage, it is the direct and only etiological factor in an easily recognized ailment which is generally medical, but sometimes surgical.

Embryology. This will be discussed very briefly. In the period from the fourth to the tenth weeks, the entire intestine composed of the abdominal portion of the foregut, the midgut and the hindgut, develops, passes out of the abdomen into the umbilical cord, evolves into its various forms and returns into the abdomen and is attached to the posterior abdominal wall in the position it will occupy ever afterwards. The colon is the last to return; the small intestines literally push the colon into position and fusion of the mesentery with the peritoneum of the posterior abdominal wall, with disappearance of the peritoneum, is the process of attachment. The right colon, being the last segment to attach permanently, would logically be the segment most liable to congenital defects. In later stages, the cecum passes from left of the midline across the upper abdomen to the region of the right kidney and finally to the right iliac fossa; in doing so, it crosses the pancreas and duodenum before reaching the kidney. Embryological membranes are absorbed and fixation as seen in the normal individual should occur; often this does not take place. The cecum may be arrested at any point in its course; this often results in the unusual positions of the cecum. Likewise, fusion of the mesentery may not be

complete and the primitive mesentery remains. This may be of any length or even to complete absence of fusion as seen in 1 patient.

The Normal Ascending Colon; Its Length, Position, Vertical and Lateral Mobility. In describing the ascending colon in this paper the cecum is considered as a part of the colon. McConnell and Hardman¹ carried out a series of studies in normal individuals and came to the following conclusions:

	Inches
Males: Ages between twenty-one and thirty-eight:	
Average length, including cecum.....	7
Average position of hepatic flexure above iliac crest, erect.....	1½
Average position of hepatic flexure above iliac crest, recumbent.....	4
Average range of vertical excursion above iliac crest, recumbent.....	2½
Females: ages between twenty and twenty-nine:	
Average length, including cecum, erect....	7
Average length, including cecum, recumbent. (Longest 9 in., shortest 6½ in., recumbent)	8
Average position of hepatic flexure above iliac crest, erect.....	¾
Average position of hepatic flexure above iliac crest, recumbent.....	3
Average range of vertical excursion above iliac crest, recumbent.....	2¾

They found that there was very little lateral mobility in 92 per cent of the patients. The colon was practically straight in 82 per cent and in 18 per cent there were one or more flexures in its course. In the males the angle of the hepatic flexure was a right angle, but in the females it was less than a right angle. The diameter of the cecum was generally that of the ascending colon. In a series² of studies of about 300 normal adults, both male and female, white and black, all

* Read before the Southern Surgical Association, White Sulphur Springs, Virginia, Dec. 11-13, 1928.

being students in local colleges, the figures given by McConnell and Hardman correspond to our findings very closely. In our series, every patient who was suffering from indigestion had a mobile cecum; this may have been only incidental, but it was very striking.

The size of the colon varies with the weight of the individual; as a rule, the cecum and ascending colon are larger than the transverse and descending colon. Space does not permit a description of the different varieties of the ptosed colon.

ORGANS AFFECTED BY RIGHT COLOPTOSIS

1. *The cecum and ascending colon* are here treated as one structure. If in 80 per cent³ of all individuals the cecum and ascending colon are securely attached to the posterior abdominal wall, rather loosely it is true, but with definite limitation of mobility, then it is reasonable to surmise that any deviation from this relatively fixed position might cause deleterious changes in function. Throughout the evolutionary ancestral ages those individuals with normally attached colons, not being subject to stasis and its allied evil effects, naturally have had a higher survival value.

The persistence of the primitive mesentery permits the cecum and ascending colon to lie lower in the abdomen and being continuous with a fixed distal ileum kinking or angulation may occur and acting through long periods of time obvious derangement in function of the ileocecal valve occurs. This may take the form either of incompetency or spasticity and become a pathological entity. If the theory of Keith⁴ is true the nodal point with its "pace maker" in the ileum may be so deranged as to produce stasis in the cecum and ascending colon with serious results.

Mass peristalsis is supposed to be the method of propulsion of the contents of this segment of large bowel; orad peristalsis is, also, normal from the cecum to the middle of the transverse colon.

By these two different forms of peristalsis, the semisolid contents coming from the small intestine are retained in this short segment of the colon until they are rendered into practically formed feces. It seems from the study of a number of case histories that there is a period during which the neuromuscular mechanism of the ascending colon is able to function satisfactorily notwithstanding the gross handicap of disturbed function due to mechanical overload. This period apparently ends from the fifteenth to the twentieth year in most individuals. Should a wasting disease, a serious acute illness or any excessive mental or psychic strain supervene, the "breaking point" may appear at any period without relation to age; or, in a few fortunate individuals, the appearance of disturbed function may be long delayed, even into middle age or beyond. Stasis is a logical consequence of disturbed peristalsis; this may be of greatly varying degrees, the absorption of pathological products from the colon as a result of this stasis is supposed to be the cause of the general constitutional maladies so frequently noticed in patients subject to this ailment.

It is a patent fact that the long-continued weight in the ascending colon is an unnatural condition. Just how disturbing this added weight is to the normal muscular functions of the colon is a factor quite difficult to measure accurately. Waugh⁵ considers this as the major etiological factor; Tyrrell Gray⁶ believes that the normal function of the sympathetic nerves, by overstimulation, becomes deranged and that this is the cause of the dysfunction of the bowel. The right flexure of the colon is always an angle sufficient to produce some measure of obstruction; in the normally attached colon, i.e., in the 80 per cent, this obstruction is physiologically overcome.

In the ptosed colon the right flexure is rarely intact; in some instances, the right flexure is in perfect form, but the whole ascending and the right half of the trans-

versed colon are ptosed. Patients with this condition do not seem to suffer from stasis as much as when the right flexure is absent. Probably this is due to a better physiological function as when once the fecal mass has passed the flexure there is less likelihood that it will exert mechanical back pressure on the mass in the segment of gut corresponding to the ascending colon.

When the right flexure is absent, there is a long uphill grade which must be overcome before back pressure is obviated. It would seem reasonable to predict a back pressure in the whole length of the colon from the cecum to the splenic, i.e., left flexure, which long continued would sooner or later force the ileocecal valve and produce ileal stasis. As evidence corroborative of this hypothesis, ileal stasis of varying degrees has been found present in this type of patient without exception and only in occasional instances in those patients with a ptosed right flexure. This type of patient is prone to have a widely dilated and greatly thinned colonic wall. The normal haustrations are frequently absent or show as slight bulging in the radiogram of the colon. Often, the distal ileum is greatly dilated and the musculature is quite thin and translucent. All of these findings are evidently the result of a long-continued mechanical pressure within the lumen of the bowel which encourages, if it does not directly cause, devascularization of the tissues of the bowel wall, producing severe tension on the sympathetic and parasympathetic nerves, both afferent and efferent, and creating pathological results in all of the other tissues of the mesentery and bowel wall. Devitalization of these structures with profound abnormalities in function are a natural and final result. Among these may be mentioned abdominal pain, tenderness, a feeling of weight, obscure nervousness, indigestion, constipation, etc., so characteristic of these patients.

2. *The duodenum* is in a position of great importance in patients suffering from ptosed right colon. The fixation of the duodenum occurs in the sixth week and

the development of the liver and pancreas further strengthens the attachment to the posterior abdominal wall. When the intestines return to the abdomen about the tenth week, the mesentery of the jejunum and ileum becomes attached to the posterior abdominal wall with the superior mesenteric artery within the stalk of the mesentery passing across the third segment of the duodenum. In the normal individual the duodenum passes beneath the mesenteric stalk without any marked compression of its lumen; but when the mesentery is dragged upon by ptosis of either the large or small intestines, narrowing of the lumen of the duodenum results. The degree of constriction depends upon the amount of pressure the superior mesenteric artery exerts on the duodenum. In the normal subject, the pressure is negligible. As a result of this pressure, which has existed from birth, there is gradually developed a recognizable change in the diameter of the gut. It is not necessary here to go into detail except to state that the final result is dilatation of the duodenum above the constricting stalk of the mesentery followed by a series of slowly developing symptoms recognized as being due to duodenal ileus.

The size of the duodenum in some instances is very great. I have seen the duodenum almost the size of the stomach and be mistaken for it during an operation. In 3 patients the duodenum was 4 inches in diameter and the pylorus had entirely disappeared, the stomach and duodenum had become a long pouch extending from the cardia to the crossing of the superior mesenteric artery.

The ptosed colon, through the ileocolic and right colic arteries, exercises a pull on the stalk of the mesentery which compresses the duodenum. The ileocolic segment is always loosely attached in coloptosis and this segment of bowel when dropped into the pelvis drags directly on the mesentery predominantly through its vascular structures. When the small intestines are ptosed into the pelvis as a

result of a loosely attached mesentery, the tug upon the mesenteric stalk produces the same compression on the duodenum as the ptosed colon; this condition is occasionally observed in conjunction with right coloptosis. When present, duodeno-jejunosomy in addition to colopexy is necessary to relieve the patient.

3. *The pylorus, the first segment of the duodenum*, or both, are dragged upon and distorted when congenital bands persist between the transverse colon and these structures. This condition is not rare in combination with ptosed colon; constriction, angulation and torsion of the duodenum may result. It is this type of abnormality which frequently simulated the symptomatology of chronic ulceration in this segment of bowel.

4. *The gall bladder* develops within the primitive ventral mesentery and as it grows to full stature congenital bands connecting the gall bladder, duodenum and transverse colon may not be absorbed; and these may persist in the adult as well defined membranes which, when dragged upon by the ptosed colon can and do produce pathological changes in the gall bladder and derangement in its functions. These may take the form of a tug on the gall bladder when the membrane is so attached that its maximum tension is on the fundus; or, a constriction of the cystic duct if the bands are so situated as to produce torsion or constriction. Acting through long periods of time the pathological result may be quite serious to the organs involved.

5. *The ileum* is always dragged out of its normal position and frequently angulated by the pelvic position which the ptosed caecum assumes, generally the ileum has a mesentery of varying length with its greatest length at the ileocaecal junction. This mobility favors stagnation of its contents and consequent ileal stasis. Various bands and membranes, congenital in origin, are found in this region. They are of slight significance, except when they cause serious angulation.

NERVOUS THEORY OF STASIS DUE TO PTOSIS OF THE RIGHT COLON

This theory was brought forward by Tyrrell Gray.⁶ He postulates the pathological consequences of right coloptosis on the theory that prolonged tension on the sympathetic nervous system, afferent and efferent, in the mesentery of the colon causes a vicious circle due to overaction of the muscles in the intestinal wall which eventually ends in dilatation and relaxation. This causes, first, deranged peristalsis as a result of sympathetic stimulation long continued; and second, weakening of the intestinal musculature which eventuates in gross dilatation of the involved section of bowel. Stagnation of bowel contents is an inevitable result followed by all the morbid consequences of stasis. Tyrrell Gray believes that all the symptoms complained of are the result of sympathetic overstimulation. The vagus supplies the motor stimulus to the entire small intestines down to and including the terminal ileum; the motor stimulus to the large intestine is supplied by the sacral parasympathetic. The sympathetic supplies the entire intestinal tract with inhibitory fibers from about the midgastric region to the terminal colon and, also, strange as it may seem, motor fibers to the pylorus and the ileocecal valve, the only two active sphincters of the bowel distal to the cardiac end of the stomach except the anus. Prolonged sympathetic stimulation, therefore, relaxes the whole intestinal tract except the sphincters which it stimulates and is the prime causal factor in producing stasis with all its supposed evil consequences. In the final stages, the sphincters, also, relax. Lane⁷ taught very forcibly the pathological effects of prolonged intestinal stasis, but his theories of aetiology are in all probability quite untenable.

MECHANICAL THEORY OF STASIS DUE TO PTOSIS OF THE RIGHT COLON

The Mechanical Theory. This theory is promulgated and championed by G. E. Waugh.⁵ In brief, he holds that the ptosed

colon by mechanical interference with the physiological propulsion of the bowel contents along the intestinal tract results

the right and left flexures of the colon, the ileocecal region, etc., is a second factor and stasis the third. These acting in con-

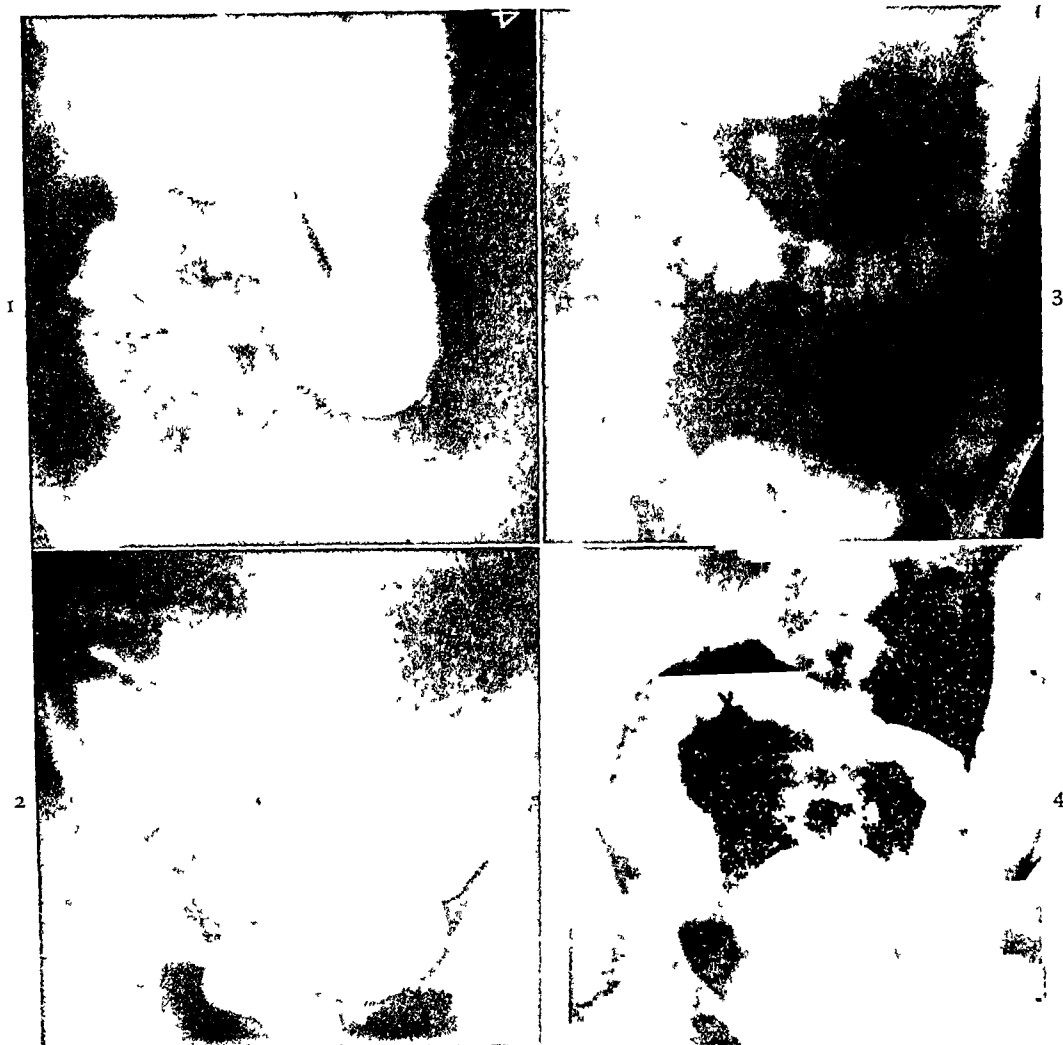


FIG. 1. Dilated duodenum which extends to crossing of superior mesenteric artery.

FIG. 2. Position of right colon in dorsal recumbent position.

FIG. 3. Right colon in standing position.

FIG. 4. Position of right colon in dorsal recumbent position, postoperative.

in a long train of morbid consequences. Waugh presents this theory in a wonderfully convincing article and fortifies his conclusions by an elaborate report of many operations. This theory is predicated upon the defects of the evolution of the human being after he changed from the horizontal to the upright posture. Gravity is assumed to be the main causative factor; disordered peristalsis, due to defective and insufficient peritoneal fixation at certain points, i.e.,

junction produce the symptoms of the typical case of right coloptosis. The mechanical theory postulates a congenital defect in the mesenteric fixation of the colon and this defect interferes with the normal mechanical functions of the bowel wall. This theory is very attractive, but probably the propulsion of bowel contents along the intestine is quite a complicated process and can not be explained so simply.

Condition of the Colon in Ptosis. Three types have been met with, viz:

1. *Spastic Colon.* The colon is smaller than normal, contracted, the musculature is thickened and the haustrations small. Colitis is always a marked feature and the patients are nervous and neurotic.

2. *Dilated Colon.* This type is characterized by the greatly dilated colon, with thinned musculature and large haustrations. The colon is often of enormous size and not the subject of marked colitis. Stasis is generally marked; the ileocecal valve is more often dilated and defective which permits the passage of gas and liquid feces into the ascending colon without the usual valvular control; ileal stasis is, also, frequently associated.

3. *The Normal Colon.* This is most often in the younger patients or in subjects with minor degrees of ptosis. Stasis is not a major factor, but if treatment is not undertaken, it may become so.

CLASSIFICATIONS OF THE DIFFERENT FORMS OF DISEASE DUE TO RIGHT COLOPTOSIS

It should now be recalled that the morbid consequences of ptosis of the right colon are anatomically limited to viscera situated in the epigastrium and the right side of the abdomen except in unusual instances. The proximal colon is the last segment of the whole intestinal tract to secure permanent fixation to the abdominal wall; and, further, this peculiar method and form of attachment is the result of the assumption of the upright posture. Also, that the attachments of the proximal colon from the cecum to about the mid transverse colon are superimposed on to other organs or interfere with their function if abnormal. For instance, the hepatic (right) flexure is attached to the right kidney or its fascia; the proximal segment of the transverse colon is normally lying on and often attached to the second segment of the duodenum; the arteries, veins and nerves of the cecum and ascending colon in conjunction with the superior mesenteric artery pass over and often

constrict the third duodenal segment; all these occur when the parietal and visceral attachments of the colon are normal, i.e.,



FIG. 5. Right colon in standing position, postoperative

in about 80 per cent of all patients. In 20 per cent, approximately, there is some degree of abnormal mobility of the right colon due to lack of fusion of the primitive mesentery with fixation to the posterior abdominal wall. The degree of mobility is proportionate to the failure of fusion of the primitive mesentery; the extent of mobility is closely related to the pathological consequences of such mobility.

GENERAL SYMPTOMS

Almost without exception, these patients are chronic sufferers from indigestion, flatulence, constipation, lassitude, mental and physical inertia as a result of long-continued intestinal stasis. They are generally neurotic dyspeptics and have run the gauntlet of the internist, the food faddist, the health resort, and if not early in their course then later, the appendix has been removed for chronic appendicitis. This usually begins a surgical pilgrimage. Chronic right-sided abdominal pain is the most disturbing complaint.

So long as the ptosed colon functions properly there are no symptoms referable

to it; it is when stasis supervenes that symptoms arise, these are general and special; while these overlap in a sense, the

SPECIAL SYMPTOMS

1. *Pain in the Right Lower Abdominal Quadrant as the Chief Symptom.* The pain

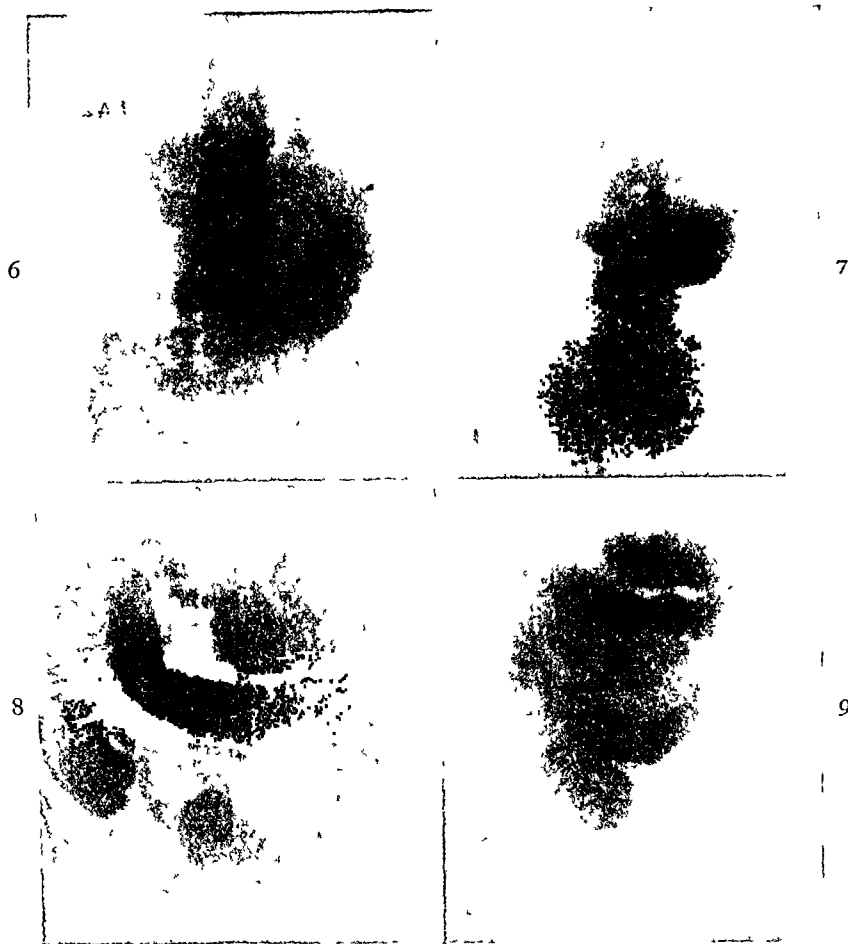


FIG. 6. Dorsal recumbent position, cecum and colon in pelvis (poor picture).

FIG. 7. Ascending and transverse colon in pelvis (poor picture).

FIG. 8. Postoperative, dorsal recumbent position, showing results following duodenojejunostomy and colopecty. Contrast this with Figure 6.

FIG. 9. Postoperative, standing. Contrast this with Figure 7.

symptoms referable to special organs are quite definite and characteristic and the approximate time of their onset can be determined with a fair degree of accuracy. Colopectosis *per se* is not a disease; but ptosis plus stasis becomes a pathological entity which not only causes general symptoms but special symptoms due to the added traction upon the nerves, membranes and blood vessels. In women, the victims of right colopectosis, dysmenorrhea, amenorrhea, menstrual irregularities, etc., are too frequent to be merely incidental.

is of an irregular, inconstant type and rarely severe; it is chronic and recurrent and generally more of a dull aching, often dragging, worse as the day passes, only occasionally felt during the night, often relieved by lying down or by taking a brisk laxative; while it may be felt after meals, the character and amount of food does not materially alter it. Prolonged, hard work, mental strain or worry, psychic storms, and constipation increase the pain. Pain may be absent for days or persist for weeks, but a chief characteristic is its variableness in intensity and duration. Tenderness over

the painful area is often present, but never as great as in inflammatory affections of the appendix or gall bladder.



FIG. 10.

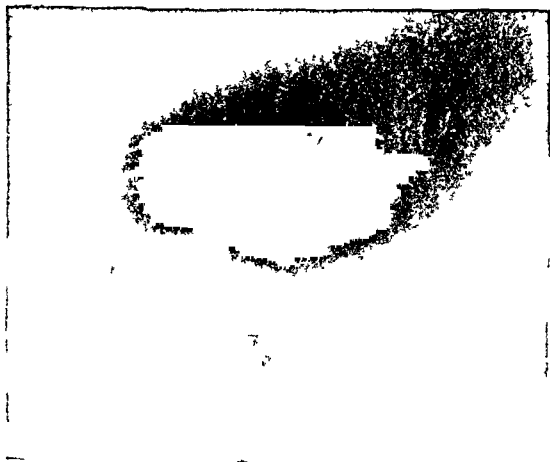


FIG. 11

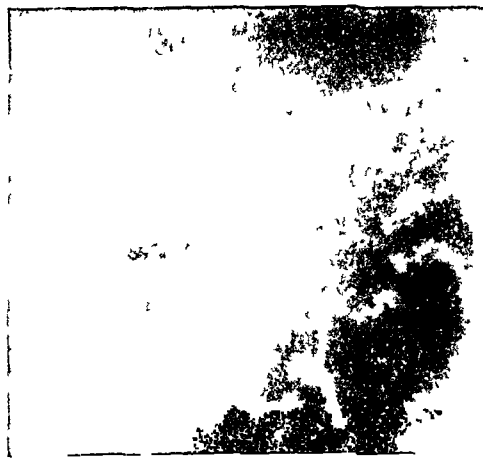


FIG. 12.

FIGS. 10-12 Case of dilated duodenum due to constriction of superior mesenteric artery as result of right coloptosis

Two of my patients were so acutely ill that the family physician brought them in with a diagnosis of acute appendicitis. Both had increased pulse rate, one a temperature of 99° , the other 100° , the leucocyte count was increased and the differential ratio raised; there was nausea but no vomiting. Local tenderness was negligible. The history was true to form and coloptosis was diagnosed; both had only slightly congested, but not inflamed appendices. The symptoms of "chronic appendicitis," so called, closely resemble those of the right iliac fossa type. This probably explains why appendectomy for chronic

appendicitis results in such a large percentage of unrelieved patients.

2. *Pain in the Epigastric Region as the Chief Symptom.* The pain in this region is, also, very irregular in manifestation. It may simulate the pain of gastric or duodenal ulcer, but rarely the former. It is seldom severe, is never paroxysmal and but little affected by the kind or amount of the previous meal. It comes on from one to several hours after meals and generally after the patient is up and about, more often in the late afternoon. There is never any periodicity or certainty of onset nor duration. Nausea and vomiting are almost never co-associated and the attack may be prolonged but rarely serious enough to keep the patient from

work. The onset of epigastric pain is synchronous with the filling of the right colon from the ileum.

3. *Pain in the Region of the Gall Bladder as the Chief Symptom.* This form is seldom seen and is generally more of an aching and is not radiated to the back nor to the right shoulder blade unless cholecystitis supervenes. In those patients who have congenital bands between the colon, duodenum and gall bladder very acute pain may be present with all the symptoms of a blocked cystic duct; the pain then is distinctly localized in the gall bladder region and there may be local tenderness

and moderate jaundice. The irregular chronicity, the absence of severe pain, especially colic, the failure of jaundice to appear and the concurrent symptoms of colon drag readily betray the true condition. However, I suspect many gall bladders have been removed for just such condition.

4. *Pain in the Right Kidney Region as the Chief Symptom.* This is the least frequent of all forms. The same irregularity of the pain which is located in the region of the right kidney is almost diagnostic. In 2 patients only have I met with kidney pain as the only symptom. It was associated with an enlarged, prolapsed, tender kidney in both instances which was made worse by the wearing of kidney pads. This can be explained on the theory the higher the kidney was raised the greater would be the strain on fascial attachments to the colon. However, Tyrrell Gray says that the pain in prolapsed kidney is due to strain on the sympathetic nerves, while Waugh theorizes that it is due to venous congestion as a result of the drag downwards by the colon.

There is often frequent urination with greatly increased amount; in some instances there is pyelitis especially in cases suffering from colitis as a complication of coloptosis.

Neurosis Associated with Right Coloptosis. As is often noted, many patients suffering from ptosis are neurotics in some degree. The question can be classified into two major sections, viz:

1. Neurotic individuals whose histories show that they were so affected before the onset of stasis; these uniformly get more pronounced unless relieved by treatment; complete cure is the exception.

2. Patients who were normal before stasis from coloptosis supervened; these patients are easily and permanently relieved by treatment.

The patient who is suffering from coloptosis is nearly always more or less neurotic. The evil effects of stasis, whether ileal, cecal or colonic produce an instability

of the nervous system eventuating in psychasthenia or some allied neurosis which is very characteristic and easily recognized. Unfortunately, all forms of neurosis have too often been diagnosed as diseases and not symptom complexes which they probably are. Improvement in the mental and psychic condition is one of the frequent early results of surgical treatment of the ptosed right colon.

MAJOR COMPLICATIONS

1. Colitis. This should be mentioned as the most important complication as it is the most frequent and the most difficult to relieve. This should be treated according to the accepted methods.

2. Pyelitis. Like colitis, this should be diagnosed before operation and then treated by the usual methods.

3. Indigestion. This is often reflex and is relieved by the colon operation; when due to some essential gastric disease, it should be treated accordingly.

4. Constipation. This becomes a minor matter if the constipation is not rectal. As the colon regains its health, constipation is relieved. If it is rectal this should be treated by the accepted methods.

5. Neurosis. This is a great problem only in those who are congenital neurotics; no form of treatment promises much. In those patients who become nervous after the onset of stasis, the operation of colopexy will often secure relief without further medication.

6. Diarrhea is a symptom in a small percentage and is often associated with rather severe colitis and achlorhydria; these two conditions are rather frequently co-associated.

TREATMENT OF RIGHT COLOPTOSIS

In approximately 20 per cent of all individuals there is a primitive mesentery of the right colon. Among this large number only a very small percentage develop symptoms. The same principles are applicable in this condition as in any other congenital anomaly. When once the colon

fails to function normally symptoms are noticed. Generally, so long as the individual is perfectly healthy otherwise, there is no disturbance in colon function; but when, because of a severe acute illness, some wasting disease, an unusual nervous or psychic strain, the colon has an added strain imposed upon it, stasis supervenes, followed by a symptom complex which is easily recognized.

The treatment is clearly medical in the early cases; surgical followed by medical treatment in the later stages.⁸ Medical treatment is so well known that it will not be discussed, except to say that many early patients are relieved especially if orthopedic appliances and physiotherapy are used with it. Surgical treatment is summed up in the word colopexy in some form suitable to the individual patient. I prefer the Waugh technique. The sole object is to restore the displaced colon to the normal position as represented in about 80 per cent of individuals.

CASE REPORTS

CASE I. Miss A., twenty-seven years old, a school teacher, weighs 94 lbs. She was a weakly, subnormal child and suffered constantly from digestive disturbances. She has been badly constipated ever since she can remember, but has never taken much laxative medicine. Formerly, she has had many "bilious" attacks during which she would have nausea and vomiting. When seventeen years old, she began to have "painful right abdomen" which has bothered her ever since. Occasionally, she would have rather severe pains, especially when she was badly constipated which would be relieved always with a brisk laxative. These attacks were followed always by extreme soreness. Finally, she had constant soreness, irregular dragging pains in the right side, her digestion became upset and she became very nervous and weak.

In 1920, she consulted me for operation as a diagnosis of recurrent appendicitis had been made. I refused to remove the appendix. Early in 1921, the appendix was removed by another surgeon; she felt better for a few months and then all of her old symptoms returned. In 1924, she consulted my associate

complaining as she did prior to the removal of the appendix. A gastroenterological study was done and a diagnosis of stasis with ptosis of the right colon and duodenal obstruction was made. An operation was advised, but refused. She gradually got worse and by the summer of 1926, she was a nervous wreck, constipated; the bowels would only move from laxatives or enemas. She suffered from pain in the right abdomen more or less constantly and complained of tenderness. The skin became pasty, muddy and pale. She had, by that time, developed a marked introspective psychosis. She did badly all summer. I examined her in August and recommended an operation which advice was not taken. She began her school work in September, but was unable to continue. She became more introspective and developed suicidal ideas and on one occasion attempted suicide. Her weight at that time had gone down to 88 lbs.

On Nov. 19, 1926, a colopexy (Waugh technique) was done. A recent letter states that she is now teaching to full capacity, has daily bowel movements, good digestion and weighs more than she has ever weighed. She feels perfectly well and has lost all of her despondent tendencies. (See Figs. 1-5.)

CASE II. Male, thirty-nine years old, book-keeper. He was a normal boy in every way and had no serious illness of any kind. When he was about twenty-five years of age, he began to have recurrent attacks of what is usually diagnosed as "sick headache." His mother suffered all of her life from the same type of attacks. These spells were limited to pain in the right half of the head, felt most acutely above the right eye. He would have to leave off his work; the attacks would last from a few hours to one or two days and would always end following a severe vomiting spell during which he would vomit a great deal of bile. He noted this and would relieve himself by producing severe vomiting.

At first, the attacks would come on any time of the day, but during the last year or so, they came on during the afternoon. Otherwise, he has been a healthy man until recently when he developed attacks of acute abdominal pain associated with tenderness in the region of the appendix which were diagnosed acute appendicitis. He came to me for operation; it was no trouble to diagnose two separate conditions,

viz: the so-called "sick headaches" and recurrent attacks of mild, acute appendicitis. A gastroenterological study disclosed marked ptosis of the right colon with extensive duodenal dilatation. Constipation has not been a marked feature of his case, although, he would have recurrent periods of constipation. His digestion has always been fairly good until during the past year when he has had some digestive troubles. The attacks of "sick headache" could never be traced to any dietary indiscretion. Operation was advised, but it was declined. His condition continued on with recurrent attacks of symptoms which sug-

gested mild, acute appendicitis, but his attacks of headache became very frequent and much more severe, coming often from one to three times a month and lasting from a few hours to several days before getting relief. He was treated for this condition, but without success.

In October, 1928, a colopexy (Vaugh technique) with a duodenojejunosomy was done. The convalescence was normal and from that date until the present; he has had no further trouble. A recent letter states that his health is excellent, the bowels are regular, the digestion is good and he has not had a single attack of "sick headache." (See Figs. 6-9.)

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TREATMENT OF GONORRHEA*

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IN this paper I will confine myself to a cursory discussion of the treatment of gonorrhea.

DIAGNOSIS. I have myself laid particular stress on the importance of accuracy in diagnosis. In making a diagnosis we make use of slides, as is commonly done, but particularly in my own department we use cultural methods. I think that very often cultures will help to give a clear diagnosis when slides are faulty. As regards complement fixation tests, personally I am a strong advocate of complementary fixation. In my own department the pathologist has by careful technique gotten something over 80 per cent of positive reactions in acute cases, which is pretty good for a complement fixation test.

Another point concerns the difficulty of diagnosis in chronic cases. I always wash out the anterior urethra well before taking a specimen of the urine or a prostatic specimen. I have often seen physicians take a specimen of urine, and then immediately take a prostatic specimen; I think in that way they often may pick up pus cells from the imperfectly washed urethra.

TREATMENT. On the whole, I have come to the conclusion that the treatment of gonorrhea is summed up in drainage and increasing the resistance of the patient.

Another school believes in the direct attack by means of chemicals: silver compounds, mercurochrome, etc. Personally I do not believe there is anything that will reach down to and destroy the gonococcus, and that ultimately the treatment comes down to drainage and increasing the resistance of the patient.

I have been interested in a new silver preparation which is used in combination with drainage. A combination of citrate of silver and nitrogen is produced, which is not yet on the market, that is deeply penetrating, and more so on account of the

hydrogen ion. We tried it, but I am afraid it is going the way of the other preparations.

Then, as an example of the fact that we are dependent on the resistance of the patient, I would draw your attention to the difference in the behavior of different cases—how one case will clear up in a few days or a week or two and others will resist treatment for a long while; and the case which has a violent reaction often clears up better than one which dribbles on from day to day and from week to week.

I had a rather interesting experience during the war. I had read a paper from America in which some one said that intramuscular injection of cyanide of mercury brought about drying-up in about ten days. I took this up very enthusiastically, and had some encouragement, for these cases did dry up very quickly. Another officer became quite enthusiastic, but he found that although the discharge seemed to dry up fairly quickly there remained a gleet which persisted longer than the usual case. I even went to the length of having new mercurial compounds made, and we tried these mercurial injections on upward of 7000 cases; but when we reckoned up we had not made a single cure. They dribbled on instead of being acute, and I really believe we came to the conclusion that the action of the mercury was to knock the gonococcus on the head and prevent the reaction, and I think one should not do this. It is very often a blessing in disguise.

As regards treatment of complications we have found heat very useful in complications of gonorrhea, and particularly in prostatic complications. I use it in my own department, applying diathermy to the prostate and vesicles in cases of gonorrheal arthritis. We simply go to the focus of infection, and find it does very well. We have no use for dia-

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thermy applied to the urethra as a direct attack on the gonococcus. I do not believe that diathermy acts by destroying the gonococcus by heat, but possibly by promoting drainage from the vesicles, and possibly by autogenic vaccination. Our experience in the treatment of diathermy applied to gonorrhoea in the female has not been favorable. We have taken cultures in cases where diathermy had been tried over a long period. Personally I do not think that diathermy does anything by destroying the gonococcus; but it is an exceedingly valuable method of treatment in such cases as gonorrheal arthritis depending on a particular focus. We also found that true of diathermy in the treatment of other complications.

The use of mercurochrome I have found quite successful in the treatment of vulvovaginitis in little girls. Some time ago on looking over the problem of vaginitis I thought of all the forms of treatment that had been applied, and came to the conclusion that most of them failed to reach the gonococcus. I could imagine it as simply laughing at us. I finally came to the conclusion that I would go for the gonococcus under visual direction. So I distended the vagina of the children, using an ordinary urethroscope, and under vision painted the fornices and a block of the vagina with mercurochrome. The first 2 or 3 cases were brilliantly successful. After that we had a number of failures; but it is quite valuable where the disease has not ascended the cervix. It is interesting to check the number of parts that one can reach by just painting, by looking to see how much has been missed. We think we have painted the vagina thoroughly, and then under vision we see it is only partly painted. Distend the vagina, look, paint again and again, and do not be satisfied until it is done thoroughly. If this is done faithfully, in a number of cases you will achieve success.

One other point should be emphasized,

gonorrhea mainly as a public health problem. I think it is the biggest public health problem of the venereal diseases. In my own country I do not believe we should be satisfied with merely the disappearance of shreds from the urine and from the prostate after previous urination. This may not be done in the United States, but in my own country it is only too common; so I spend a good deal of my time preaching for very thorough cure; for it is only thus that one can eventually get the better of this problem.

We are often asked what we achieve by all this elaboration of treatment. Often I have been told by my superior officer that "I don't think you get any better results with your new fangled treatment than we got in the old days. We got ours well in six weeks." I generally reply respectfully: "Yes, but they were back again promptly." Now, since we have introduced better methods of cure I am convinced that the number of relapses is infinitesimal; but the treatment of gonorrhea is a difficult problem, a more difficult one than the problem of syphilis, and it is still far from solved.

RESEARCH. What line of research would be a helpful one? I think we are going to solve this problem by proving the value of autogenic vaccine. You may be interested in a new type of vaccine elaborated at . . . my old hospital. There the pathologist has found a certain number of strains (gonococci grown on a special medium) and is able to separate these toxins from the rest, and applies this toxin by injecting into the urethra, and also intradermally. The results so far have been very promising, but I think it is a work that will take a good deal of time and energy. But I will make a suggestion. I think myself that we shall not advance very far by means of chemotherapy or any vegetable organism, but I do think we shall achieve something by improving the value of autogenic vaccine.

INTESTINAL OBSTRUCTION*

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MY discussion will be limited to acute intestinal obstruction. Little progress was made in the treatment of this condition up to five years ago, notwithstanding the great improvements in other fields of surgery. Since the epoch-making work of Orr and Hayden¹ on blood chemistry in relation to acute intestinal obstruction chief interest has centered in the toxemia associated with obstruction and a more rational basis has been found for treatment of this serious condition. The most fatal cases are those where obstruction occurs high up and the toxins are most fatal in the duodenum. There has been much discussion as to the nature of these toxins: are they bacterial in origin, or are they protein bodies?

B. W. Williams² of London, has pointed out the importance of toxemia due to anaerobes in acute obstruction and peritonitis. The late symptoms of peritonitis are identical with those found in fatal cases of intestinal obstruction. Adynamic ileus is the result of peritonitis and the general manifestations of fatal cases of peritonitis are identical with those of the terminal stages of intestinal obstruction. The chief symptoms in common are rapid pulse, cyanosis, slight general icterus, and especially restlessness and a pathologically acute consciousness up to the very end. Williams points out what was observed by army surgeons: that these symptoms bear a striking resemblance to those observed in severe cases of gas gangrene. This led to investigations to determine the part played in acute obstruction by anaerobic organisms. The one most commonly found is bacillus Welchii. This organism is constantly present in the intestines and produces a very powerful toxin. The organism grows best in a slightly neutral medium such as is

found in the duodenum. In acute obstruction and late peritonitis there is great proliferation of the B. Welchii.

Williams has been using an antitoxin prepared from this organism at St. Thomas Hospital in London for two and one-half years in acute obstruction and peritonitis associated with paralytic ileus. Only the most severe cases were treated; the series consisted of 256 consecutive and unselected cases; there were only 3 deaths in the series. This is indeed a remarkable showing in a type of case where the usual mortality is about 50 per cent.

This new knowledge together with that made available by Orr and Hayden, in their experimental work on dogs, I believe has laid the foundation for a revolution in our treatment of acute intestinal obstruction. The salient facts in Orr and Hayden's work are (1) the diminished chlorides in the blood, (2) the increase in non-protein nitrogen, (3) increased carbon-dioxide combining power of the blood. Orr also showed experimentally that a restoration of the normal chlorides greatly prolonged the life of dogs with intestinal obstruction, and that jejunostomy hastened death. Dogs with intestinal obstruction live longer than those with a simple high jejunostomy. The life of dogs with high obstruction was greatly prolonged by the administration of chlorides. In view of this experimental evidence and collected statistics in human beings, the value of jejunostomy in intestinal obstruction is of doubtful value. The mere opening of a loop of intestines in no way insures drainage. Peristalsis is essential for this process. The loops of obstructed and paralyzed bowel hang like wet rags over a rope, only stripping of each individual loop will adequately empty the bowel of its toxic material.

This brings us to the consideration of a

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more thorough method of operating in early cases of obstruction. Dr. W. B. Holden of Portland, Oregon advocates complete eventration of the acutely obstructed bowels and the introduction of a large glass tube, secured in the bowel by a phlange around which a catgut suture is tied. All obstructed loops are then quickly emptied by stripping between two fingers of the gloved and vaselined hand. He has reported a series of over 100 cases in which the mortality has been reduced to 20 per cent in the early cases.

With all of the foregoing facts in our mind let us outline the course to be followed by the surgeon in these acute cases of obstruction.

1. The cases diagnosed early and operated upon within twenty-four hours do not wait for blood chemistry examinations nor for roentgen-ray findings. Open the abdomen through a lower midline incision and seek for the site of obstruction. If it is a band, a twist, or an intussusception that is easily relieved and the bowel is in active peristalsis, no more need be done. If there are many water-logged loops of bowel eventrate on a hot towel, quickly empty them of their contents, restore them to the abdominal cavity and close without drainage. Either simultaneously with the operation or immediately following give 2000 c.c. of normal saline subcutaneously.

2. Cases which have been obstructed more than twenty-four hours are usually toxic. Immediate operation is often contraindicated. The surgeon is justified in taking time for a blood chemistry and restoration of the chlorides in the blood. To this end while waiting for the blood

chemistry report 500 c.c. of 3 per cent normal salt solution should be administered by hypodermoclysis and the stomach washed out. In view of the results obtained by Williams in the use of the bacillus Welchii serum, if obtainable, its administration should be begun to counteract the effect of the toxins. Operation should not be performed until the chemical balance in the blood has been largely restored.

Immediate operation releasing the obstruction will only permit a lethal dose of the toxins to escape into the undamaged bowel where it will be quickly absorbed. As soon as the blood chemistry approaches normal under the continued administration of salt solution, operation should be undertaken to remove the obstructing lesion, or if necessary, resect the gangrenous bowel. The success of operation in these advanced cases depends upon speed and accuracy and provision should always be made for the escape of gases through a catheter introduced into the bowel above the site of obstruction, in such a manner that it can be removed without reopening the wound.

The old dictum: operate immediately in acute intestinal obstruction, should no longer be followed unqualifiedly. Each case must be analyzed and the treatment adapted to the individual case. Already the beneficial effects of the new knowledge are being reflected in the mortality statistics.

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SPINAL ANESTHESIA*

OBSERVATIONS BY A GENERAL ANESTHETIST: CONSTANT
PERCENTAGE FACTORS

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I N the entire field of general surgery and its associated branches, during the decade since the World War, greater progress has been made in the specialty of anesthesia than in any other surgical department. This period has witnessed a marked development in the perfection of gas-oxygen apparatus; ethylene has been introduced and found its place; ethyl chloride, as an induction agent for ether anesthetics, has become more adaptable and more acceptable; carbon dioxide and oxygen therapy have made tremendous strides, and many hydrocarbon compounds have become the subject of extensive experimentation, each seeking its particular level. However, especially during the last two years, the alert and aggressive student of surgical literature cannot help but be impressed with the interest being displayed in spinal anesthesia (or rather analgesia) as a means of alleviating the pain of surgical procedures. This revival of enthusiasm dates from the epochal disclosures of Pitkin,⁶ concerning his researches which led to the development of spinocain, (the solutions of both heavy and of light specific gravity) and to his improvements in technique which finally have made spinal anesthesia controllable, accurately scientific, and safe.

The general anesthetist should not be too tardy in adopting this method of anesthesia and adding it to his repertoire, and it is proposed to discuss the reasons for the general anesthetist becoming proficient in this particular art, along with such observations on technique as necessarily must be somewhat different when the spinal anesthesia is administered for the "other fellow."

This manuscript will not concern itself with the fundamental principles of controllable spinal anesthesia, as these have been pointed out so admirably by Pitkin in his many writings. Neither will it elaborate on lumbar puncture technique and spinal anesthesia in general, since these features are covered in the recent literature by Labat,⁵ Babcock,¹ Koster,⁴ and others, as well as by the standard texts on surgery and surgical technique. However, from a background of considerably more than 10,000 recorded, personally administered anesthetics (general anesthetics), with practically every well-known and reasonably applicable anesthetic agent, covering a period of more than ten years of specialization in anesthesia, and from an intensive study and experience in spinal anesthesia according to the Pitkin technique, I wish to present certain observations which should be of primary and fundamental interest to anesthetists, as well as to those engaged in the practice of general surgery.

Ether anesthesia, since its introduction by Long, eighty odd years ago, has been a wonderful boon to humanity. And it still is. However, it has certain elemental drawbacks, as all alert surgeons and wide-awake anesthetists recognize. Primarily, it is the surgeon's anesthetic but not the patient's, because it gives to the surgeon the possibilities of extreme relaxation, leaving the patient to bear the brunt of its after effects, such as they may be, even when given expertly. Gas-oxygen anesthesia and gas-oxygen combined with field-block have been of remarkable advantage in certain types of surgery, for with this form or combination, the patient

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may be given a most pleasing anesthetic with a minimum of disagreeable post-operative after effects, and the surgeon can secure by proper cooperation, in most instances, the desired relaxation necessary for his mechanical procedures. However, in spinal anesthesia, or rather more scientifically analgesia, we have a method which promises to prove a most ideal anesthetic from the viewpoint of *both the surgeon and the patient* for all surgery below the nipple line. It can be administered painlessly and has no after effects whatsoever; it is non-toxic. It gives the surgeon that marvelously soft and quiet abdomen, or relaxation of the skeletal muscles in the case of surgery on the lower limbs, which facilitates to a remarkable degree the ease with which he can work out the mechanical features necessary to the performance of the required operation.

General anesthetists throughout the country, if they wish to keep abreast of the times, surely must qualify themselves to administer spinal anesthesia. The method is here to stay. It is reasonable and rational; it is accurate and scientific; it is controllable and safe.

During recent years, an anesthetic-consciousness has been awakened among the general public; they have acquired an increased knowledge of medical and surgical affairs; they are asking for better anesthetics than in years past; the cruder and more antiquated methods of yesteryear must give way to the more modern and the more scientific measures of today. The American public, through the world's most extensive and most highly developed mediums of publicity (newspapers, magazines, and radios) not only has become more travel-minded, more motor-minded, and more air-minded, it also has been educated to be more medical-minded. Anesthetists of today are better trained; the public demands it. Hospitals must meet the very highest standards; the public demands such institutions and has a right to have them. Diagnosis and treatment of disease must be modern and scientific; the public demands that this

be so and it is entitled to the best. Anesthetists of today must be willing to sacrifice time, effort and money in attending clinics where they can study and work out this problem of spinal anesthesia. It is not a method to be acquired merely by a casual consideration of the subject and a hurried reading of the technique. Nor should it be attempted by those whose brain cells are inactive, atrophied or inert. Mental alertness is a prerequisite, as is also a sound and basic training in surgical technique, because, to administer spinal anesthesia successfully, experience must be developed to the nth degree, almost to the point of artistry. There is no such thing as half-results; either one has it or he hasn't; either he knows it or he doesn't. Half-measures most certainly will subject the anesthetist to the odium of defeat, besides placing the patient in grave danger. One must use judgment. Judgment is knowledge plus experience. Knowledge can be acquired by a study and a review of the text and the literature, and by visiting our modern clinics. Experience is gained by observation and practice. The anesthetist should go where he can follow closely one who is skilled in the method of spinal anesthesia; then, with articulated spine or cadaver, practice and practice and practice again. That is the way to acquire a *beginning* of judgment. This must be followed by keeping accurate records of all the concomitant features of each case. A study of these records will help to mature judgment and give that sound and basic knowledge of the art which is so highly essential to success. Intensive concentration on failures, some of which inevitably will occur among early cases, will be far more instructive than will be successes.

In every surgical center, many advantages accrue by the presence of a skilled anesthetist capable of administering spinal anesthesia. For example, one anesthetist can take care of the surgical requirements of several busy surgeons, or of quite a few more men who operate only occasionally. The busy surgeon, one who operates

several or more times daily, does not have the time to devote to the acquiring of the technique of spinal anesthesia, when there is present a skilled anesthetist capable of doing it and thus saving many minutes of the surgeon's valuable hour, or hours. Nor, on a busy morning, can he afford to spend the necessary minutes used in preparing each succeeding case. The occasional surgeon, one who operates but once or twice a week, does not have a sufficient number of cases whereby he can master the fundamentals and gain enough experience to become proficient in the art. Further, the surgeon is interested primarily in solving the patient's pathological problem and seeing the patient get well; anesthesia, while a most necessary accessory to the result, is a field by itself. Hence, it is the province of the specialist in anesthesia; he must come through. As another example of how the anesthetist can save a busy surgeon's time (and wear and tear on his nervous system): Case I in the morning is prepared by the anesthetist by the time the surgeon arrives on the operating floor; Case II is made ready while the surgeon is finishing Case I. The surgeon can step immediately to the neighboring operating room and operate on Case II. While he finishes Case II, Case III is anesthetized for him by his anesthetist, and so on. Where a routine such as this is adopted, it is surprising and almost remarkable how much valuable time is saved for the surgeon who actually is interested in his own efficiency.

From my records, I detail briefly an instance of this character: June 3, 1929, 3 consecutive cases for one surgeon: The first was listed to begin at 9 A.M. Spinal tap was made for Case I, and the solution injected, between 9 and 9:05. The operation was uterine dilatation and curettage, high amputation and repair of the cervix, uterine suspension, and appendectomy. Case II was prepared toward the close of the first, this operation being appendectomy plus a general pelvic and abdominal exploration thoroughly made *with complete manual and visual inspection due to the relaxed musculature*. Case III was

prepared while the skin sutures were being placed in Case II; operation: cholecystectomy and appendectomy. This third patient was on the cart on her way back to her room at 11:10, the total elapsed time for the three operations, from the very start to the complete finish, being two hours and ten minutes. We were not pressed for time; no effort had been made to hurry; in fact, haste was not even thought of; it merely was one of our average, routine performances. This demonstrates most conclusively that spinal anesthesia, criticized by some as a time-consuming procedure, actually can be made a time-saving element for the busy team, especially as the marvelous relaxation so greatly facilitates the speed with which the surgeon can accomplish his ends. Naturally, speed presupposes a surgeon who actually can go ahead with judgment and dispatch, easily and rapidly, in contrast to the occasional surgeon whose efforts generally use up many more of the valuable minutes.

In administering spinal anesthesia for the "other fellow," there are certain points of technique which differ materially from instances in which the surgeon induces the anesthesia for himself; for upon the anesthetist's head, and his head alone, will fall the not too softly padded club of disapprobation should the resultant anesthesia be anything but a success. Surgeons are *entitled* to good anesthesia; patients *demand* it.

Many surgical cases are presented to the anesthetist for anesthesia without a complete disclosure from the surgeon (perhaps unavoidably so) of what he fully intends to do; this not always because of a lack of study of the problem but often on account of the intricacies involved, as we all well know. For example, the surgeon may state that the operation is a simple appendectomy; yet, before he is through, he may go up and take out the gall bladder, or at least he may palpate in the region of the gall bladder. Now, if the block with spinal anesthesia, in a case such as that outlined, extends only as high as midway

between the umbilicus and the ensiform, the patient will complain of pain and will not permit palpation of the gall bladder or its removal, or even a general exploration of the upper abdominal cavity. Then one must resort to adjunctive anesthesia by gas-oxygen or ether, and both the surgeon and the patient will heap direst calumny on the head of the anesthetist and label the anesthesia a failure. To prevent such disaster and to forestall all such criticism, it has been my policy in abdominal surgery to block all cases as high as the nipple line; and, where I know for a certainty that high abdominal surgery is going to be performed, I block these cases to a point midway between the nipples and clavicles, not being particularly worried if the anesthesia *on the surface of the body* extends as high as the clavicles themselves.

This may seem a rather bold and rash procedure; but, if the reader will study with me a few of the basically anatomical and interrelated physiological points involved, I believe he will agree that it is not as radical as it seems. *Under proper observation and technique, it is reasonable, rational and safe.*

In many instances, to my direct knowledge, a remark common among surgeons, even by very excellent and quite worthy surgeons, when discussing spinal anesthesia, is: "I don't mind seeing the anesthesia go as high as the umbilicus or even close to the rib margin, but when it gets as high as the nipples I'm worried about the diaphragm and the respiration." Let us examine this and see whether there is justification in their fears. The major muscles of respiration are: the diaphragm, the levatores costarum, the infracostals, and the triangularis sterni. The accessory muscles are five: quadratus lumborum, pectorales, sternomastoid, infrahyoid, serratus magnus, and the extensors of the spine. This latter group may be dismissed because of its *relative* unimportance, i.e. their action is not so marked. The *motor nerve supply* for the first group is as follows:

Levatores costarum	} Anterior primary divisions of the intercostal nerves derived from the dorsal cord.
Infracostals	
Triangularis sterni	
Diaphragm	} Phrenic nerve and, slightly, the diaphragmatic plexus of the sympathetic.

Keep in mind two other points, namely. First, the phrenic nerve is derived mainly from the fourth cervical segment of the cord, with branches from the third. Secondly, the cervical plexus supplies the surface of the body to a point just below the clavicles. Accordingly, spinal anesthesia, as tested by a forceps on the skin, as high as a point midway between the clavicles and the nipple line means that the anesthetic solution has not ascended higher than the first dorsal segment of the cord. The cervical cord and cervical plexus are not involved; the phrenic nerve, the chief motor supply to the diaphragm, is not paralyzed. It is true that abdominal respiration is lessened, markedly so in anesthesia of this height, due to paralysis of the abdominal and costal muscles; but, by close observation of the epigastrium, one will always see some movement there transmitted to it by the diaphragm.

Koster⁴ says: "Interruption of conductivity always comes quite some time before the excitability is lost," and "sensory impulses enroute to the cerebrum may be stopped in the medulla because conductivity is interrupted, whereas the cardiac and respiratory nerve centers in the medulla, although their excitability is lowered, can still respond to the physico-chemical stimuli furnished by the blood by the initiation of motor impulses." If this is true, we should not be afraid even though dilute mixtures of our anesthetic solution reach the cervical segments of the cord. However, I do not wish to infer that one should be careless. This is no method for the inexpert. These patients must be watched. Alertness in

observations, keenness in judgment as to dosage, and perfection in technique are prime requisites.

Pallor or cyanosis always are worrisome and troublesome features, when present, both to surgeon and to anesthetist, in *any* form of anesthesia. However, in my spinal anesthetics, I seldom encounter them, due, I believe, to the fact that I counterbalance my splanchnic dilatation with sufficient ephedrine. I routinely administer 100 mg. of ephedrine along with the preliminary injection in all abdominal cases; and, if the anesthesia inadvertantly extends above the point of midway between the nipple line and the clavicles, I frequently give from 50 to 100 mg. additional. These doses are well within the limits of toxicity and produce happy results. Oxygen and carbon dioxide also are beneficent adjunctives. No conscientious anesthetist ever is justified in giving any anesthetic without having available, for almost instant use, a liberal supply of oxygen and carbon dioxide certainly not in our modern hospitals. Patients go there to be surrounded by every safeguard, and they deserve to have this one. It is surprising, at some clinics, both those in which spinal and general anesthesia are being used, what little attention is paid to the phenomena of pallor and cyanosis. It seems that the anesthetist and surgeon go blissfully and serenely on, ignorant or indifferent to the impending situation. Pallor and cyanosis mean but one thing to me—sub-oxygenation. This can be due to several causes, including surgical shock and hemorrhage, either of which may demand immediate attention; but, in most cases, it is the result of respiratory depression, central or peripheral. In general anesthesia, over-morphinization or over-anesthetic dosage may bring it on; in spinal anesthesia, paralysis (partial or complete) of the diaphragm, intercostal muscles, and other accessory respiratory muscles may be the causative factors. If, in spinal anesthesia, the depression is central, i.e. the medulla, then the

anesthesia has extended *too high*, greatly beyond the realms of necessity. In *either event*, oxygen and carbon dioxide are strongly indicated: oxygen to oxygenate the blood and tissues, carbon dioxide because it is our most potent (besides being physiologically correct) direct stimulant to the respiratory center of the brain. The patient's response to this therapy is almost incredible: pinkness replaces pallor and cyanosis; the respiration is almost instantly increased in rate and amplitude; the nurses breathe a sigh of relief; the anesthetist quits perspiring; the surgeon drops his stare of dismay and goes to work.

To produce satisfactory spinal anesthesia properly to the nipple line or to the clavicles in most instances requires a slight modification of Pitkin's technique as to dosage of spinocain and the necessary resultant expansion with spinal fluid. To use but 2 c.c. of spinocain expanded to 8 c.c. total will not give anesthesia of this type; or, if it does, the anesthesia will be slow in coming on (twenty minutes or more) and transitory in its effect, or will demand marked variations in the plane of the Trendelenburg position, or it may require a higher point of injection, and, usually, it is more fluctuating in degree of intensity of anesthesia. It may not last long enough to complete such major surgery as requires an hour or longer, sometimes much less. Even 3 c.c. are insufficient in some cases for anesthesia of this height; 4 c.c. often must be used. Four cubic centimeters of spinocain expanded to a total of 8 c.c. with spinal fluid gives a 5 per cent mixture to inject into the subarachnoid space, which percentage in my clinical experience, approaches the ideal strength solution for optimum results. I admit that this is 400 mgm. of novocaine, but I have seen no operative or postoperative reasons to believe that it is an excessive dose. On the other hand, I have witnessed many very very happy results from this technique. If it is true, as published in

the literature,⁶ that 100 per cent solutions of novocaine injected into the spinal canals of dogs produce no inflammatory reaction two, three, and four days later, and, if it is also true, as we well know, that a 40 per cent solution (of small volume) can be used most satisfactorily in obstetrical anesthesia, then I can see no reason why a 5 per cent solution should be objectionable for routine use. Further, if to produce anesthesia of the legs only, 2 c.c. of spinocain expanded to 4 c.c. (now 5 per cent) are injected, why not maintain the same correlation when desiring 8 c.c. total expansion for gall-bladder surgery? Clinical experience leads me to believe that a fixed coefficient will give more uniform success, certainly in average cases.

Novocaine in the subdural space is practically non-toxic. I have used 600 to 1200 mg., (i.e. 12 c.c. of spinocain) in one patient without untoward results, operatively or postoperatively, details of which case will be given later.

However, the on-coming anesthesia with these higher percentages must be observed exceedingly closely, so that it does not extend too high. These patients must be watched; amateurs should not attempt the technique until they have gained proficiency. While the dosage in volume must be varied for different types of patients and cases, the percentage mixture should remain the same; nothing could be more reasonable. A "constant percentage factor," such as this 5 per cent standard mixture, should be of essential importance in teaching spinal anesthesia, remembering that the height (extent) of anesthesia is governed by volume injected and that the intensity (duration) depends upon the percentage strength. Future clinical experience may prove that a constant percentage factor of 4 or 6, or even 8 or 10, will be more desirable, but some uniform equation must be used to attain uniform results. At present, I am using the factor 5 with most beneficent success.

The degree of Trendelenburg position

must be watched; it may be necessary to keep the patient level for a short period until the anesthesia becomes fixed before lowering the head 5 or 10 degrees. One must be on the lookout for short spines, long spines, large (around) spines, small spines, stiff spines, flexible spines, scoliosis, lordosis and kyphosis. One must be sure that the degree of inclination of the spine and of the table correspond, i.e. that the spine is straight instead of being cocked up at one end or the other. We have had 1 case, giving a history of spinal injury during childhood, in which we roentgenographed the spine preoperatively to make sure that lumbar puncture could be performed satisfactorily; with this patient, any other course would have led to defeat. One should ascertain beforehand from the surgeon what he expects to do. One must use a little of his own judgment as to how long each particular surgeon *may* take! In using the word "occasional surgeon" in this manuscript, no disrespect is meant to the men who operate only once per week; many of them do good and basically sound surgery. However, it must be recognized that, through lack of organized effort of the team and less dexterity in handling surgical instruments, their surgery often is more prolonged than that of surgeons who operate more frequently.

CASE I. May 11, 1929, female, thirty-four years old, married, height 5 ft. 5 in., weight 190 lb. Diagnosis: acute appendicitis; surgeon, occasional. From the history, I suspected an additional chronic salpingitis; and, suspecting that a long time might be needed, I injected $4\frac{1}{2}$ c.c. of spinocain expanded to 9 c.c. total, giving anesthesia to the supraclavicular region. Enormous pus tubes were found, one the size of a large grapefruit, the other only slightly smaller. The operation lasted two and a half hours. No supplemental anesthesia was used. Recovery was uneventful, even beautiful.

CASE II. May 8, 1929, female, thirty-seven years old, married, height 5 ft. 6 in., weight 250 lb.; operation: appendectomy; surgeon, occasional. Five cubic centimeters of spinocain

expanded to a total of 10 c.c. were used, giving anesthesia to the neck. Operation lasted one and a half hours. Anesthesia was satisfactory in every respect, recovery uneventful.

CASE III. May 10, 1929, male, thirty-seven years old, height 5 ft. 10 in., weight 240 lb.; operation: appendectomy; surgeon, occasional. Five cubic centimeters of spinocain, expanded to 10 c.c. total, were used. Anesthesia was given to clavicles, satisfactory to everybody concerned. Recovery was uneventful.

These 3 cases were unusual and not at all easy from any viewpoint, yet they are just as likely to turn up in anybody's practice as they did in mine (and may in the future). The technique, briefly stated, shows what can be done once the anesthetist has developed his judgment to the point of knowing just exactly what he is doing to the most minute detail. Any other course would be folly! Each of these patients had morphine $\frac{1}{4}$ grain and 100 mg. ephedrine preoperatively. Pulse, blood pressure and color remained satisfactory throughout. To have piddled along with a mere 200 or 300 mg. of novocaine in these cases would have been to invite disaster, as far as the anesthesia lasting long enough is concerned, and bring down upon spinal anesthesia, both from the surgeon and from the patient, that peculiarly significant odium called failure.

It may be true, as some authorities state,⁴ that 200 mg. of pure novocaine crystals *dissolved in 8 c.c. of spinal fluid* will produce analgesia of the entire body, but in spinocain we have a solution which by its specific gravity and by its viscosity entirely changes the complexion of the situation. It is necessary to understand the hydrodynamics of the spinal fluid and its circulation. The specific gravities of the solutions used must be constantly before you. The fundamental laws covering diffusion and dissemination, fluidity and viscosity, and absorption and volume control must be carefully studied.

Other known facts led me to develop the technique of injecting a 5 per cent solution of novocaine in my spinal anesthetics, i.e.

when I wish a total expansion of 8 c.c. I use 4 c.c. of spinocain, when I wish expansion of 6 c.c. I use 3 c.c. of spinocain, and so on. First, Babcock¹ states that an 8 per cent concentration of novocaine is necessary for effective anesthesia; I use a 5 per cent maximum with very happy results, remaining well within the limits of the aforementioned dosage. Secondly, the protopathic sense (pain) is lost earlier and more completely than is the epicritic sense (touch), and the nerve roots first reached are affected most intensively and for the longest period. Now, with the weaker dilutions, patients may keep the sense of touch (even after pain is abolished) and then, hearing the instruments click, they are afraid of being hurt and will complain, causing the surgeon to look at the anesthetist in disgust and casting suspicion on the method of anesthesia. These same phenomena occur when the anesthesia is dissipated before completion of the surgery and often are the cause for a need of supplemental anesthesia. I have seen this repeatedly when using dosages of less concentration; and, if the patients have any sensation whatsoever, there always are those only too ready to criticize, to scoff, or to sneer. The solution is obvious. Thirdly, one wants the anesthesia to come on relatively quickly, not only because this saves the surgeon's valuable time but also because the anesthesia will be better, i.e. it will last longer and the relaxation will be more complete. Fourthly, the posterior roots of the spinal nerves control pain sense, tractile sense, temperature sense, and muscle sense.¹ The anterior roots control voluntary movement, muscle tonicity, and the superficial and deep reflexes. It is evident from a study of these facts, remembering that spinocain is lighter in specific gravity than is spinal fluid, that its viscosity delays dissemination, and that the patient will be lying supine on the table, that more spinocain, plus a regular and a constant percentage factor for expansion, are required to obtain maximum results. A knowledge of the

differences in conductivity between the anterior and posterior roots of the spinal nerves is of the most importance when using heavy novocaine solutions in spinal anesthesia versus spinocain. A discussion of these latter features I wish to reserve for a future manuscript.

Another point in favor of blocking high with spinal anesthesia for abdominal surgery is as follows: Supposing a simple appendectomy is about to be performed. Anesthesia as high as the umbilicus will permit the incision to be made painlessly, but only the lower half of the rectus and transversalis muscles will be blocked and the relaxation of the abdominal wall will not be as complete as if the entire rectus is paralyzed. The patients will complain that they feel what is being done. Traction on the cecum will give referred pain in the chest (generally interpreted by the patient as about the heart) due to the sympathetics not being blocked high enough. The rectus abdominis and transversalis muscles receive their nerve supply from the spinal nerves of the lower six dorsal segments of the cord, consequently it is necessary to allow the anesthetic solution to run *at least* that high in the subarachnoid space of the spinal canal before good anesthesia will result.

Still further facts constantly to keep in mind are that the segments of the cord do not coincide in location with the corresponding vertebra of the spinal canal and that the innervation from each spinal nerve is not horizontal but tends downward, points of great value if one wishes constantly to visualize in his own mind just exactly what is taking place within the spinal canal. And this must be done to win success.

A factor recurring so frequently in my clinical experience with spinal anesthesia as to convince me of its reasonableness as a clinical fact is that the "prematurely gray" individual is more resistant to novocaine solutions within the spinal canal than are normal patients or those who are normally gray for their age.

Recognizing the difficulty of determining "norms," by the prematurely gray individual I mean a person of twenty-five years to thirty-five years of age (or thereabouts) whose normally blond or brunette hair is fairly well interspersed with white or gray, as one would expect of an individual of forty-five years to fifty years of age. I have had to repeat my injection on so many of this type of patients (even with my 5 per cent constant factor solution) that I have come to regard those whom I do not reinject as the exceptions to the rule. In the normally gray, those whose grayness corresponds with their years, this difference does not seem to obtain. The difference, too, has been more noticeable when using the regular spinocain than when using spinocain "heavy," probably due to the latter's higher concentration on account of lesser expansion, as will be presented later.

CASE IV. A female, single, thirty-eight years old, rather nervous type, iron-gray hair, medium build, not obese, weighing 140 lb.; operation: cholecystectomy and appendectomy. She had a preliminary hypodermic of morphine $\frac{1}{4}$ grain. Her systolic blood pressure was 240. When I made the spinal tap, in the second lumbar interspace, I found the fluid under considerable pressure and allowed 30 drops to escape, by which time the rate of dropping was lessened perceptibly. Being the third patient of this type which I had met within a short period of time, I determined upon what I considered to be a large dose, 5 c.c. of spinocain. This I expanded nicely and easily with spinal fluid to a total of 10 c.c. After injection, I kept her lying on her left side with spinal column and table perfectly level, watching and testing closely. Recognizing in ten minutes that the anesthesia was incomplete, I made a second tap, again nicely and easily, and gave 3 c.c. spinocain expanded to 6 c.c. After again waiting ten minutes, I made a third tap and gave 4 c.c. spinocain, unexpanded, and in a few minutes had the necessary anesthesia. I had given 100 mg. ephedrine in the preliminary novocaine solution.

Here, then, was a patient who received 12 c.c. spinocain, i.e. 1200 mg. or 18 grains

of novocaine, yet she caused no operative or postoperative concern. She had no pallor or cyanosis; her pulse throughout was excellent; one hour and thirty minutes after the final injection she could move her legs and all sensations had returned. Apparently, the dosage was not too large for this particular individual for, after obtaining anesthesia, the effect lasted only normally long.

I do not wish to infer that all prematurely gray individuals should be given this large dosage, but this (plus previous and subsequent experience) gives me more confidence in the non-toxicity of spinocain in case a second or third injection is needed in the event that the standard dose is insufficient, and it influences me not to hesitate to repeat my injection (after proper interval) in any patient (especially of this type) if I watch my on-coming anesthesia closely enough and understand the chemiophysiology involved.

What is the explanation? Nerve fibers consist of an axis cylinder, a sheath of fatty myelin, and a thin covering called the neurolemma.³ Within the spinal canal the nerve roots are in relation with the meninges of the cord, each having a covering of pia mater, and the arachnoid membrane covers each root. It is my opinion that in prematurely gray individuals (certainly in a considerable percentage of them) these vestures of the nerve roots are more difficult to penetrate by the spinocain solutions.

It is hoped that this only too brief and necessarily intimate discussion of some of the concomitant problems involved in spinal anesthesia will stimulate and encourage the general anesthetist to study and become proficient in the art. It may help the general surgeon to appreciate some of the intricacies which his anesthetist is endeavoring to solve in order to safeguard the life and welfare of the patient.

When introducing spinal anesthesia into any given surgical center, the greatest care and judgment must be exercised;

for, often through ignorance and lack of knowledge, or because of other ulterior motives, many times the method is basely maligned before sufficient trial has been made. To administer spinal anesthesia indiscriminately, especially where other agents are indicated in preference, would be an anesthetic folly. On the other hand, to deny spinal anesthesia to those patients whose needs cry out for it is an anesthetic crime. Educational publicity of the highest ethical standards must be promulgated among both surgeons and the laity, of which, among both surgeons and laity, the best is a demand from satisfied patients and their friends. The ball is rolling.

The practice of medicine, in all its branches, is a progressive science; he who stands still for the briefest part of a decade is soon left hopelessly behind. The world moves, and we must move with it. Any hospital, or surgeon, or anesthetist, who stands obstructively upon the right of way of the on-rushing train of progress likely will be found among the debris and wreckage by the wayside. I believe that spinal anesthesia is here to stay; because, in its modern exemplification, many advantages accrue to both the patient and the surgeon. It is reasonable and rational; it is accurate and scientific; it is controllable; it is safe.

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INTERSTITIAL PREGNANCY*

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ECTOPIC pregnancy has long been a favored subject of reports and discussions among gynecologists. Numerous studies from the clinical aspect together with the gross and microscopic pathology of the condition have formed the basis for more accurate diagnosis and more efficient treatment, as well as for numerous theories to explain the cause of a fertilized ovum embedding itself in regions not suited to its complete development.

The fertilized egg may become primarily implanted in any portion of the tube, in the ovary or as now seems proved, in rare instances, on the peritoneum. The site of implantation in the tube has given rise to the anatomic classification of ampullary, isthmic and interstitial pregnancy. That portion of the tube surrounded by uterine muscle is the shortest part, the smallest in caliber, the simplest in design of its mucous membrane folds, and the least common site of a tubal conception. In spite of these facts the interstitial pregnancy has not been allowed to rest as such but has been further divided into groups. The simplest classification is tubo-interstitial, utero-interstitial, and interstitial proper. It would seem sufficient however to label any ectopic pregnancy implanted in the wall of the uterus as a true interstitial gestation. That its development may be laterally toward the isthmus or centrally toward the uterine cavity cannot be denied but the great majority develop posteriorly. The ovum must arrive at its place of settlement by ascending or descending the tube, or an accessory tube and may implant itself in the mucosa of the tube, an accessory tube, or a diverticulum of the tube. All explanations of the cause are theoretical and can be found in

any textbook of gynecology and obstetrics. The pathologic anatomy and the pathologic physiology of both the tube and the ovum may influence its ectopic implantation.

Ten years ago I reported two rather early ruptured interstitial pregnancies. Since that time I have observed and operated upon two women in whom the gestations had advanced for much longer periods. A review of the literature showed about 85 cases reported prior to 1914 which were considered proved while I found three well described cases and added two of my own. There were in addition 16 incompletely described but probable. In the last ten years I have accepted 50 more cases and found 14 inadequately described but probable. In discussing specimens presented before societies a considerable number of gynecologists have referred to cases they have seen but whether these same cases have been recorded with descriptions of the specimens is impossible to state.

Diagnosis. Before operation the accurate diagnosis has been made rarely and not often suspected. While in the great majority of cases after rupture has occurred a diagnosis of ruptured ectopic pregnancy should be obvious it is suprising how often these women have been kept under observation in a hospital for several days before operative treatment has been instituted. I believe we are justified in assuming that interstitial pregnancy is more puzzling than any other variety of ectopic gestation. Intrauterine pregnancy and threatened abortion associated with cornual myoma are especially difficult to differentiate in some instances. I finally made the correct diagnosis in one of my cases after observation for several weeks during which time the woman refused operative interference.

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The following preoperative diagnoses have been recorded in thirty-seven of the present series of 50 cases:

Unruptured interstitial pregnancy...	1
Ruptured interstitial pregnancy....	3
Interstitial pregnancy with ovarian cyst	1
Ruptured ectopic pregnancy probably interstitial...	1
Tubal pregnancy or pregnancy in uterine horn.....	1
Ruptured ectopic pregnancy	14
Ruptured ectopic pregnancy with myoma of cornu	2
Acute appendicitis or ectopic pregnancy	2
False labor with peritonitis	1
Incomplete septic abortion	1
Incomplete abortion and fibroma of cornu	1
Impending abortion with fibroid	1
Pregnancy with fibroid	1
Retroverted incarcerated pregnant uterus with undetermined complications	1
Internal hemorrhage	1
Fibroid of cornu	1
Adnexal tumor	1
Ovarian cyst	1
Pelvic inflammatory disease	1
No diagnosis	1

It is interesting that the diagnosis of pelvic inflammatory disease does not play a major rôle in the differential diagnosis as is usual in tubal pregnancies. The preoperative diagnosis depends almost entirely upon a history suggesting ectopic pregnancy and the palpation of a mass in the uterine cornu.

Total amenorrhea was noted 11 times and amenorrhea until the onset of acute pain 5 times while some irregular bleeding through the vagina occurred 17 times. Total amenorrhea is decidedly more common in this than in other types of ectopic gestation.

Pain prior to rupture has not been noted carefully in a sufficient number of reports to determine its presence, character or location. Schuman and Vineberg claim that pain before rupture is located nearer the mid-line than in the usual tubal

pregnancy. Pain after rupture does not differ from that occurring in any ruptured ectopic.

Nausea, vomiting, fainting, and collapse occur as in any severe internal hemorrhage. On examination before rupture a mass, varying in size according to the age of the pregnancy, can be felt in the uterine cornu. It is fairly firm, more or less tender and is attached to the uterus by a broad base from which it extends upward and slightly outward. After rupture the exquisite tenderness and large blood clots in the pelvis usually make a satisfactory mapping out of the pelvic viscera impossible. The accurate determination of the origins of the round and utero-ovarian ligaments and tubes is rarely possible either before or after rupture. The uterine body is said to be rather larger than in other tubal pregnancies.

The blood findings in this series have been noted infrequently. Doubtless the blood picture is similar to that of ordinary tubal pregnancy. Shortly after rupture a high leucocyte count is found while the hemoglobin and red cell count will be unexpectedly high but will drop until the low point is reached forty-eight to seventy-two hours after the hemorrhage. This was true in a series of severe hemorrhages occurring on the gynecologic service of the Johns Hopkins Hospital which I observed twelve years ago.

The pulse and temperature are of value in differentiating inflammatory lesions.

The diagnosis is not always clear at operation and a microscopic examination is at times necessary to establish the presence of a pregnancy as well as the exact location. At operation the origins of the round and utero-ovarian ligaments and of the tubes should be compared on the two sides and their relations with the pregnant horn noted. Pregnancy in a bicornate uterus or in a rudimentary horn may easily be confused with unruptured interstitial pregnancy before operation but should offer little difficulty under the eye. In general the round ligaments

will originate on the anterior and inferior surface of the sac somewhat lateral to the middle, while the tube will be lateral and inferior but higher than the round ligament. The tube and the round ligament and the utero-ovarian ligaments will be more widely separated than on the non-pregnant side.

After removal of the specimen an absolute diagnosis can be made in many cases by macroscopic sections alone but in other instances it is necessary to make a thorough microscopic study. The following points should be determined: (1) a fetus or fetal elements must be found, (2) uterine muscle must surround the fetal membranes except at the region of the rupture, (3) there must be no connection with the uterine cavity unless definite evidence of a rupture into it is found, and (4) the isthmus of the tube must not be involved in the sac. The absence of uterine glands and of a true decidua in the pregnant horn while there is a uterine decidua in which no fetal elements are found eliminates an intrauterine pregnancy or a pregnancy in a rudimentary horn. Decidua is not always found in the uterine cavity as it may be discharged after the death of the fetus, sometimes as a cast. The comparative sizes and lengths of the two tubes and sections through the tube about the junction of the isthmial and interstitial parts will eliminate the possibility of an isthmial pregnancy. The ultimate fate of the fetus is death. An early ovum may die and be absorbed. The pregnancy may become secondarily abdominal with continued development of the fetus but as far as I can find there has been no report of a living baby delivered by operation. It is extremely difficult to prove that a large fetus in the abdominal cavity had its beginning as an interstitial pregnancy.

Age. The youngest patient was eighteen and the oldest forty-four.

Years	15-20	20-25	25-30	30-35	35-40	40-45
Number	1	4	17	13	9	7

Previous Pregnancies. Thirty-four women had had one or more full-term

babies (i para 9, ii para 12, iii para 3, iv para 3, v para 4, vi para 1, vii para 1, xii para 1), 6 had one or more abortions only, 15 had one or more abortions as well as full-term babies, and 5 were never pregnant before.

Previous Operations. Several women had undergone some pelvic operation several months or years before their present illness.

Pelvic Inflammation. Previous inflammatory disease of the tubes which apparently plays an important rôle in the causation of ectopic pregnancy has been definitely noted so infrequently that the percentage for the series cannot be determined.

Occurrence. Von Rosenthal found 40 interstitial pregnancies among 1324 ectopics prior to 1896 but many of the 40 were considered extremely doubtful by Werth and Weinbrenner. In my former paper 1577 extrauterine pregnancies from 10 large clinics showed only 18 of the interstitial type (1.14 per cent).

The following series were not included in those figures.

INTERSTITIAL TYPE AMONG ECTOPICS

	Interstitial	Ectopics
Bertino	2	56
Brady	0	50
Farrar	3	309
Foshett	1	117
Frank	0	105
Grad	2	53
Grusdell	1	80
Laurantait	1	100
Levy	1	45
Monroe-Kerr	1	80
Polak	6	307
Prestatozza	2	200
von Schrenk	14	610
Wiegand	4	210
Zimmermann	2	83
Total	40	2405 (1.66 per cent)

It is interesting that in the author's series of 304 ectopics there were 2 interstitial and 2 ovarian pregnancies.

Treatment. Operation by the abdominal route is necessary. The usual methods of combating internal hemorrhage are employed. In the severe cases operations should be done with the greatest rapidity and with the least operative trauma,

coincidentally with blood transfusion or intravenous saline as may be possible. The type of operation selected depends upon the individual problem. If the pregnant sac is not too large the cornu may be excised and the wound sutured, but supravaginal hysterectomy can be done more speedily and safely in the more advanced pregnancies. Cornual excision is better and safer than curetting out the sac followed by packing or suture. Kelly opened a woman's abdomen, made the diagnosis of interstitial pregnancy and then curetted out the contents from below. Hirst and Farrar following Kelly's suggestion have performed similar operations. Kelly does not advocate this operation, and it would seem that cornual excision offers a more speedy method with less danger of serious hemorrhage and infection. Some surgeons advocate hysterectomy in every case but this view is certainly too radical. The type of operation was mentioned in 45 of our series. One woman died without operation and the specimen was obtained at autopsy.

Operation	Number Cases
Wound trimmed and sutured	1
Removal of fetal membranes with suture.	3
Excision of pregnant sac	2
Excision of cornu	10
Supravaginal hysterectomy	26
Hysterectomy	3

Seven women were given blood by transfusion.

The pregnancy occurred in the right side 29 times and in the left side 22 times, and one (Woolf's) was possibly bilateral.

Fate of the Ovum. The ovum may die and be absorbed. The fetus may die but the syncytium continue invading maternal tissues. The sac may rupture into the peritoneal cavity, into the uterus, into the broad ligament, or into the isthmus of the tube (theoretically possible but highly improbable and no such case on record). The vast majority rupture into the peritoneal cavity and occasionally a secondary abdominal pregnancy supervenes. It has been suggested that the sac may rupture

into the uterine cavity and the fetus develop to full term and be delivered spontaneously. This may be possible but no instance with adequate proofs has been reported. In this series the age of the pregnancy has been estimated 34 times.

Age, mo	1	1-2	2-3	3-4	4-5	5-6	7
Number	1	8	8	12	3	1	1

Thirty-eight were ruptured and 14 unruptured. The site of the rupture was mentioned 17 times as follows: postero-superior 7, posterior 4, superior 3, antero-superior 1, superolateral 1, lateral 1. The cases of Gilbert and Mosti ruptured into the uterine cavity but unfortunately there are no microscopic descriptions.

Prognosis. Traub in 1893, and a week later Lawson Tait, performed the first operations for interstitial pregnancy. The mortality without operation cannot be known but is undoubtedly high, due to the enormous hemorrhage which usually follows rupture. It is stated that rupture during the first three months is followed by greater blood-loss than when it occurs during a later stage. In 1918 I found 10 deaths in 82 operations (11.9 per cent). Six deaths in 47 operations are recorded in the present series. Comparing this mortality with that of a large series of operations for all types of ectopic pregnancy performed during the years 1890 to 1923 at The Johns Hopkins Hospital during which time there were 354 (Wynne's series 1890-1916, 304 cases and Brady's series 1917-1923, 50 cases) of which 13 died (3.7 per cent), it is evident that interstitial pregnancy is the most dangerous type of ectopic gestation. The uterine cornu may well be called the bloody angle.

Operation before rupture should have an extremely low mortality. After rupture occurs rapid transportation to a hospital with immediate and speedy operation utilizing the modern methods of combating serious blood loss are the only means of lowering the mortality.

In view of the diagnostic difficulties we have given the histories of our two patients in considerable detail.

CASE 1. Mrs. F., a white woman twenty-one years of age, married four years, was delivered by forceps three years ago, and the

sent to the University Hospital. The following day she was examined under anesthesia and an attempt made to lift the uterus up, but it

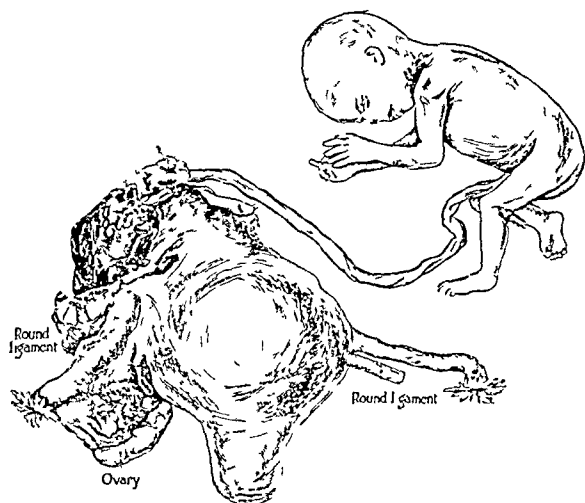


FIG. 1. Case 1. Mrs. F. Interstitial pregnancy, right. (Reconstructed drawing.)

puerperium was uneventful. Her past history was unimportant. The last menstruation was normal in every way beginning March 15, 1921. There had been no bleeding from the vagina since. On June 26, following an automobile ride, she had sudden sharp pains in the pelvis which lasted intermittently for two or three hours. She was nauseated and vomited once but felt well the following day. Two weeks later an attack of pain began which lasted a week but did not confine her to her bed. She felt well another week but following an automobile ride, the pain returned very suddenly at 11 A.M., July 26. I examined the patient an hour later in her home. She was in considerable pain and cried out at five-minute intervals. The temperature was 98.6, the pulse 72, the respiration 16. The mucous membranes were of good color, the skin was pale and moist. The breasts contained colostrum and the areolae were deeply pigmented. The abdomen was generally tender but there was no muscle spasm or rigidity, no kidney tenderness and the flanks were tympanitic. Pelvic examination showed no vaginal discharge. The cervix was high up behind the symphysis, not enlarged and rather firm. The culdesac was filled with an insensitive mass continuous with the cervix and of the consistency of a pregnant uterus. A tentative diagnosis was made of a retroverted, incarcerated, pregnant uterus with undetermined complications. The patient was

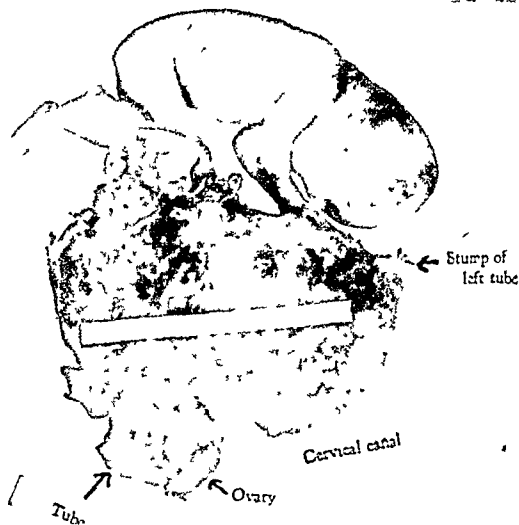


FIG. 2. Case 1. Mrs. F. Interstitial pregnancy, right. Anterior view of specimen removed at operation after fixation in 10 per cent formalin. (Photographed.)

was adherent on the right. The patient refused operation at this time. Leucocyte count 18,000, erythrocyte 3,600,000, hemoglobin 54 per cent, polymorphs 98 per cent. Ectopic pregnancy was considered but we did not feel certain of the diagnosis. The patient was quite comfortable the following two days then agreed to operation.

Operation. The abdomen was opened through the lower mid-line and a dark red tumor presented in the wound. There was a little dark, bloody fluid and a few small clots in the peritoneal cavity. The left tube and ovary were normal. The appendix showed no evidence of inflammation but a small clot was adherent to its tip. The uterus was enlarged, very soft and in the right cornu a large rent tamponed by a fetus within the unruptured membranes. There were rather firm adhesions between the pelvic wall, sigmoid, rectum, and the right uterine cornu. The right tube was increased in diameter and the right ovary surrounded by adhesions. There was considerable bleeding after adhesions were released. A supravaginal hysterectomy was performed. Convalescence was prolonged by infection in the subcutaneous fat and by secondary anemia. She was discharged from the hospital September 5.

Pathologic Report. The specimen consisted

of the body of the uterus, the right tube, and the ovary. The uterus was asymmetrically enlarged in the right cornu where there was a

thickness, infiltrated with polymorphonuclear leucocytes and small round cells. No chorionic villi were found.

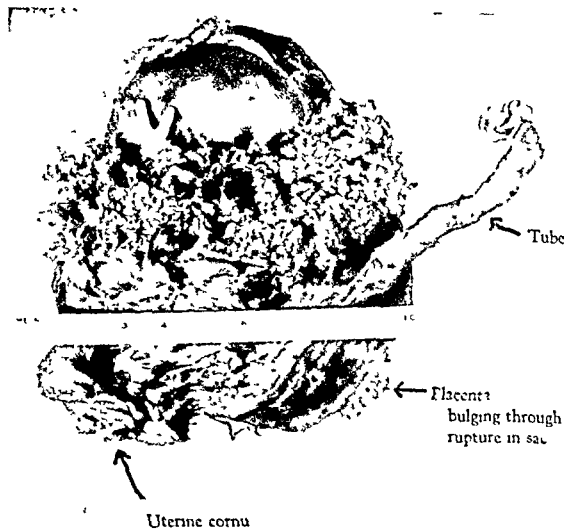


FIG. 3. Case 2. Mrs. B. Interstitial pregnancy, right. (Reconstructed drawing.)

wound on the superior-posterior surface, measuring 7 by 4 cm. with ragged edges through which placental tissue bulged. A male fetus measuring 12 cm. was attached by its umbilical cord to the tissue in the cornu. The specimen measured $8\frac{1}{4}$ cm. from the internal os to the center of the top of the body, 9 cm. between the origins of the tubes, $\frac{1}{2}$ cm. between the insertions of the left tube and left round ligament, $1\frac{1}{4}$ cm. between the insertions of the right tube and right round ligaments. The right round ligament was attached to the anteriolateral and inferior aspect of the right cornu but was 2 cm. higher than the left round ligament. The right tube was 8 cm. in length and was enlarged to about three times the normal diameter. The right ovary measured $1\frac{3}{4}$ by $\frac{3}{4}$ by $\frac{1}{2}$ cm. No connection between the uterine cavity and the gestation sac could be found. Section through the uterus lateral to the gestation sac showed a cross-section of the interstitial portion of the tube and uterine muscle. Uterine muscle could be seen surrounding the sac except over the area of rupture.

Unfortunately a careless technician lost all of the blocks and the gross specimen except one section through the uterus.

Microscopic Examination (G-21-56). Anterioposterior section through the uterine body showed endometrium 0.5 to 1 cm. in

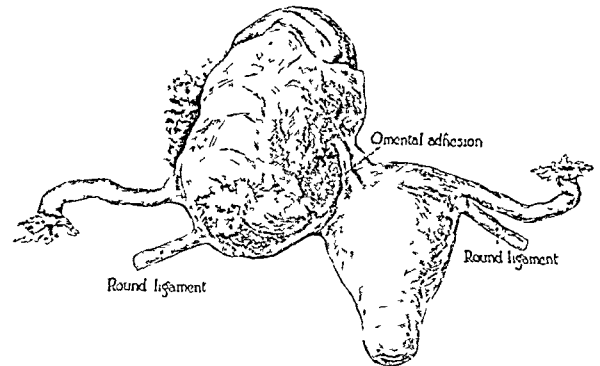


FIG. 4. Case 11. Mrs. B. Interstitial pregnancy, right. Posterior view of specimen removed at operation after fixation in 10 per cent formalin. (Photograph.)

CASE 11. Mrs. B., a white woman, thirty-three years old, married twelve years, has one child five years of age, born by normal labor with no puerperal complications, and had a drug-induced abortion at 1 month eleven years ago without complications. Her health has always been good except a slight anemia and nervousness for some years. Her last menstruation began December 29, 1921, ended January 5, 1922, and was normal in every way except for a slight increase in amount. For three weeks she had felt tired, nervous, and depressed and had morning nausea. She took some pills to induce an abortion on January 21. About 8 A.M., January 22, she got out of bed and went to the toilet where she felt a pain in her right shoulder followed shortly by abdominal cramps, chilly sensations, dizziness and a profuse sweat. I first saw her at 9 A.M. the same day, sweating profusely and a little pale. There was some general abdominal tenderness but no muscle spasm or rigidity and no tenderness in the flanks. The breasts contained no colostrum. Pelvic examination showed the cervix low, slightly enlarged, softened, bilaterally lacerated and motion caused pain in the left pelvic region. The corpus was not well outlined, the left adnexal region was slightly tender but there was no tenderness in the right fornix. Pulse 66, respirations 14, and temperature 97.8°F . The hemoglobin was 70 per cent (Sahli), red blood cells 4,320,000, leucocytes 9000. The urine was normal. A tentative diagnosis of ectopic pregnancy was made. Hospitalization was refused. At 6 P.M. the temperature was 99.4°F . Deep pressure over

any part of the abdomen caused pain in the shoulders and neck and a similar pain was caused by coughing.

February 17, she came to my office complaining of occasional slight pains in the abdomen. The uterus was asymmetrically enlarged on the right and softened, the cervix was cyanotic and soft, the fornices were not tender. The blood pressure was 116/70.

March 1 she complained of soreness at the right costal margin, general abdominal pain, nausea and vomiting, chilly sensations and nervousness. The abdomen was slightly tender over the right side, a little more marked over the right lower quadrant. There was tenderness on both sides of the uterus which had enlarged since the last examination.

March 2 the abdominal pains lasted all night. Examination revealed no change. She agreed to hospital observation but as the pain subsided quickly she left the hospital within forty-eight hours. A diagnosis of probable right interstitial pregnancy was made.

March 24 at 3 A.M. pains began in the right abdomen which were relieved by a hot water bag. At 8 A.M. she went to the toilet and the pains became severe, followed by nausea and vomiting, sweating and weakness. The abdomen was quite tender over the entire right side and flank. There was no jaundice. Pulse 70, respiration 20, temperature 97°F. At 5:30 P.M. the abdominal tenderness had increased and rigidity was present. The posterior fornix was exquisitely tender but no blood clot crepitus was made out. There was no bluish discoloration of the navel. There was a fairly firm mass to the right mid-line extending to within 3 fingerbreadths of the navel. Pulse 100, blood pressure 110/70, hemoglobin 61 per cent (Sahli), leucocyte 20,000. At no time since her period ended January 5, had there been any sign of blood from the vagina. She finally agreed to operation.

Diagnosis. Interstitial pregnancy, right, ruptured. Operation at St. Barnabas Hospital. The pelvis was full of fluid blood and there were two handfuls of clot over the right kidney. The omentum was adherent to the anterior parietal peritoneum to the left of the mid-line and to the upper surface of the pregnant sac. The right uterine cornu was enlarged to the size of a small grape fruit. The corpus was slightly enlarged. The left tube and ovary were normal. The right tube was of the same size and length as the left. The right

ovary was normal. No corpus luteum of pregnancy was identified in either ovary. The right round ligament was attached to the anterior and inferior surface of the enlarged cornu and was an inch higher than the attachment of the left round ligament. The right tube joined the cornu much higher than the left. The distance between the round ligament and the tube at the right cornu was more than twice as great as a similar measurement on the left. The right cornu was ruptured posteriorly and placental tissue was bulging through a rent $2\frac{1}{2}$ by 4 cm. through which the unruptured membranes were visible.

The right tube and uterine cornu were resected and blood removed from the abdominal cavity. The patient made a rapid and uneventful convalescence.

Specimen. The specimen measured 10 by 8 by 7 cm. The tube measured 8 cm. in length and was normal in appearance. On the posterior superior surface of the cornu there was a hole with ragged edges measuring 4 by 3 cm. and a small hole 1 cm. in diameter on the anterior surface just above the insertion of the round ligament. Placental tissue bulged through both ruptured areas and through the larger opening the unruptured membranes enclosing the fetus could be seen. The area where the cornu was amputated from the uterine body measured 4 by 5 cm. The tube left the sac 3 cm. above and lateral to the stump of the round ligament. The fetus was a female of 14.5 cm. crown-rump measurement.

Microscopic Examination (G-22-38). Section through the wall of the sac showed uterine muscle with syncytial cells invading the inner muscle layers. Chorionic villi were present and there was a spotty decidual reaction.

Sections through the isthmus and ampulla of the right tube showed no evidence of inflammation, no chorionic villi and no decidual reaction.

Sections through the uterine muscle of the right cornu including the tip of the right horn of the uterine cavity showed thickened endometrium with enlarged interstitial cells and some small round cell infiltration. The glands were about normal in size. There were no chorionic villi. Sections through the interstitial portion of the tube lateral to the gestation sac showed no chorionic villi.

I wish to express my thanks to Dr. J. C. Litzenberg, Chief of the Department of

Obstetrics and Gynecology of the University of Minnesota, for his kindness in

supplying the drawings, photographs and microscopic sections of the specimens.

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INDUCTION OF LABOR BY RUPTURE OF THE MEMBRANES AND ADMINISTRATION OF PITUITARY EXTRACT*

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INDUCTION of labor by artificial methods is now recognized as a necessary procedure in many conditions arising in the practice of modern obstetrics. The induction may be for the benefit of the mother, the baby, or both.

This subject is not new, as it was reported in 1789 by Denman, an obstetric physician, to the Middlesex Hospital, London; that as early as 1756, a number of eminent physicians met together in London to consider the treatment of labor as complicated by contracted pelvis, with special reference to the moral rectitude and advantages of the early induction of labor. After due deliberation this procedure was given their general approbation. Dr. Denman, in reporting a case of contracted pelvis, in which he had performed the operation three times, resulting in two living children, gives no minute instructions as to the method employed, but apparently he knew of none besides that of rupture of the membranes.

The next contribution to the subject is found in "Medical Facts and Observations of the Year 1800," in a letter of Mr. John Barlow, a surgeon of Lancashire. In this letter, disclosing a form of treatment which he employed in cases of contracted pelvis, he wrote as follows: "Upon my plan of delivery the life of the child is not endangered while the mother sustains no greater risk than that incurred in natural labor. And I will venture further to advance that by the method I am about to describe the formidable obstetric apparatus of knives, hooks, and perforators may be happily banished in the future from our surgery." This new plan was the induction of premature labor by rupture

of the membranes during the seventh month of gestation.

Improved surgical technique has so increased the comparative safety of the cesarean section that what was apparently the sole indication of the past for early induction of labor (viz., extreme contracted pelvis) has now ceased to be a factor in this department of obstetrics, because of the greater risk to the child from prematurity.

We, however, with the advance in prenatal care, have increased the indications for premature induction of labor in such of our cases as present: slightly contracted pelves near term; antepartum bleeding; toxemia; eclampsia; cardiac disease; renal complications; the birth of successive dead babies at term without apparent cause; hydramnios; discomfort near term and so on for almost without number. A study of the procedures now advanced for inducing labor seems to show that none are universally satisfactory to the physician or the patient, nor are they always certain of accomplishing the desired result with safety and without delay. In the beginning, I will review the methods most commonly used at the present time. These divide themselves into two classes of treatment: (A) medical, and (B) surgical.

A. *Medical.* Under the first class we find ourselves dealing principally with various methods of the administration of castor oil, quinine, and pituitary extract given subcutaneously or by absorption from mucous membranes. These are augmented by further stimulation from hot baths and warm enemas.

At term it is claimed these methods result in the onset of labor in from 50 per

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cent to 90 per cent of the cases, depending on the vigor and persistence with which the method is pursued.

It is to be remembered that the more remote the date of expectancy, the greater the proportion of failures to be expected from the administration of medicinal agents.

From both the accoucheur's and the patient's viewpoint, if a start results it is highly satisfactory because both feel that no real interference with nature's methods has taken place. If no result ensues, which on the law of chances, all cases both slightly premature and at term being considered, is a probability in from at least 40 per cent to 50 per cent of attempts, the patient has had a very uncomfortable twelve to eighteen hours. Yet many women in their desire to have the pregnancy completed are willing to repeat this dosage two or three times.

These methods are valuable, however, and should be tried and retried in cases where it is desirable, but not necessary, for the patient at or beyond her due date to be started in labor.

If the institution of labor is essential and delay dangerous, medical methods should be augmented by other procedures unless they give immediate results.

B. Surgical Treatment. In general it must be stated that under no condition should the induction of labor by surgical procedures through the vaginal route be attempted unless the operator feels secure in the fact that delivery may be safely completed by that route, viz., the compatibility between the passenger and the passage must be complete.

Under surgical methods of induction of labor we find ourselves dealing with three fundamental lines of procedure: (1) Accouchement forcé. (2) Methods dependent on the insertion of a foreign body into the uterus, which acting as an irritant institutes uterine contractions. (3) Induction of labor by rupture of the amniotic sac and augmented by the use of pituitary extract.

These we will consider in some detail.

1. *Accouchement forcé.* Forced manual or instrumental dilatation of the cervix, with the extraction of the fetus following, is in my mind to be avoided at all times, save in cases where the cervix is entirely thinned out, and the os at least partially dilated. Even under these conditions, the method is to be pursued only when immediate delivery is imperative.

The use of powerful mechanical dilators of the Bossi type is universally considered to have little or no place in present day obstetrics.

All cases forcefully dilated are likely, on inspection, to show excessive tears of the vagina, cervix, and lower uterine segment, and are not infrequently accompanied by grave shock and severe hemorrhage.

In cases which offer insistent indications for immediate delivery, without labor or any cervical dilatation, the obstetrician should make his choice in cesarean section of either the abdominal or the vaginal route.

2. Methods of induction of labor involving the introduction of a foreign body into the cervix or uterus to stimulate uterine contraction.

a. Probably the most popular method associated with this class of induction lies in the use of the inflated rubber bag of which several types, shapes, and sizes are available.

The use of the bag is especially to be recommended in cases of central or partial placenta previa, where delivery from below is deemed advisable. The insertion may be made intra- or extraovular. The disadvantages of the bag are:

(1) Some degree of instrumental dilatation may be necessary for the insertion of the collapsed bag of the required size to give dilatation indicated.

(2) If preliminary dilatation is not obtained in this way, repeated insertion of bags of increasing size is necessary.

(3) Anesthesia may be required to complete the introduction.

(4) There is difficulty in maintaining

true asepsis during insertion and subsequently.

(5) Dilatation when the bag is expressed is not complete or satisfactory. The cervix is "stretched up" rather than "taken up."

(6) The presenting part is pushed out of the pelvis when the bag is filled.

(7) With extrusion of the bag the cord may prolapse and be compressed by the presenting part returning to the pelvis.

(8) In many cases we find complete failure or extremely delayed onset of labor.

b. The Krause method, viz., the introduction of a soft rubber bougie (10 to 12 mm. in diameter) as high as possible between the amniotic sac and the uterine wall. Labor ensues usually within twenty-four hours but may fail, requiring the introduction of another bougie, or recourse to some other method.

c. The third method under this class consists in packing the cervix and vagina tightly with sterile gauze. It is a method not generally satisfactory, and in the majority of cases only softens the cervix without inducing labor, so that other procedures are necessary to accomplish active uterine contractions.

d. Many other schemes have been suggested, i.e., the injection of fluid in the space between amnion and wall of the uterus, but they are so fraught with danger or are so unreliable that discussion is not indicated here.

3. The preceding pages cover briefly the methods most commonly used in the premature induction of labor, with the exception of the one which bases the stimulation of uterine contractions and subsequent labor on the rupture of the membranes and the escape of the amniotic fluid. This procedure augmented by the use of pituitary extract has been used by the author with favorable results for the last few years, in such cases as would seem benefited by early induction of labor and it is my desire to present it for your consideration.

As is shown by the letter of Dr. Denman in 1789, mentioned earlier in this paper,

rupture of the membranes to induce premature labor is not new in obstetric history. In fact, it is apparently the first method advanced for this purpose. It is without question the simplest and most natural maneuver to be considered, nature herself instituting labor in a not inconsiderable percentage of pregnancies by preliminary rupture of the amniotic sac. From the latter fact it is undoubtedly true that our predecessors of the eighteenth century took the idea directly from nature herself.

Artificial rupture of the membranes is technically easy, it is safe, and from the surgical point of view can be performed with complete cleanliness. In fact the maneuver is accompanied by little more risk than the ordinary vaginal examination during labor. Furthermore, with the membranes ruptured, the doors are not closed to the use of the other methods of labor induction already mentioned.

In cases listed at the end of this paper the routine pursued is essentially as follows:

1. The patient is prepared by the usual ether, iodine (7 per cent) or picric acid (10 per cent) followed by alcohol.

2. A long hysterectomy clamp is easily and without force passed through the os uteri until its tip encounters the fetal head. The jaws of the clamp are opened slightly and then closed while held firmly, but not forcefully against the vertex. If the tips of the clamp close accurately they will pick up the membranes, which rupture as the forcep is drawn outward. The examining fingers of the left hand are held in the vagina throughout this maneuver, acting as a guide for the forcep both in regard to its direction and its relation to the presenting part. The clamp should be long enough to permit easy manipulation by the right hand without the hand coming in contact with the vulva.

3. All the fluid that can be readily released should be encouraged to escape as the time in which labor ensues depends greatly on the proportional amount of water escaping. If the head is wedged into

the pelvis and lower uterine segment, gentle upward pressure may be used to raise the presenting part slightly, care being taken not to disengage it completely. Another maneuver that may be tried is to slip a long closed clamp behind the head, posteriorly, as one applies a blade of the obstetric forcep, and then open it a little, thus making a gutter for more fluid to run through. When sufficient fluid has escaped, the examining fingers and clamp are withdrawn. As stated above this all involves little more chance of sepsis and risk than a vaginal examination.

4. The patient is then put in the prone position and the fetal heart auscultated. Auscultation should be repeated frequently for a time to be sure the cord is not pressed on unduly.

5. In many cases the uterus will show signs of contractions almost immediately and in these cases, further stimulation is not indicated. In the cases where no activity of the uterus appears after twenty to thirty minutes, pituitary extract minims 2 to 5 is given hypodermically. It will be found that in almost all instances contractions will occur after a few minutes resulting in labor. If for any reason labor does not start within an hour, and this is usually due to the escape of too little fluid, the injection of pituitary extract may be repeated.

In concluding this paper recommending the rupture of the amniotic sac and the injection of small doses (minims 2 to 5) of pituitary extract to accomplish the premature induction of labor, I am reporting 87 cases. These results are collected, principally, from my private records, augmented by reports from the records of Dr. Raymond S. Titus, Dr. Delos J. Bristol, and from the files of the Boston Lying-In Hospital.

Of these 87 cases, 26 (29.9 per cent) were primiparae while the remaining 61 (70.1 per cent) were multiparae ranging from para ii to para xii. Eighty-nine fetal

births resulted from the 87 pregnancies (2 twin cases) with 79 babies living and well when discharged from the hospitals. The 10 cases in which the babies were not living at birth or died shortly after are accounted for as follows: 5 macerated feti; 4 premature infants (six and one-half months); and one anencephalic monster. The babies were delivered in 40 instances without operative interference; low forceps were used 33 times; midforceps, 7 times; and in the remaining 9 cases, labor was completed by internal podalic version or breech extraction.

All mothers were discharged obstetrically well.

These cases bring out interesting figures in regard to the actual labor time; the time elapsing between rupture of the membranes and delivery; and the relation of the time consumed by primiparae as compared with that of the multiparae.

The average time of actual labor for the entire series was five and four-tenths hours; the longest being twenty-four hours while the shortest interval was thirty minutes.

The time consumed from rupture of the membranes to delivery averaged eight and one-tenth hours, the longest interval being thirty-four hours and the shortest was again thirty minutes.

In the primiparous cases the approximate average time was seven and one-fourth hours of actual labor, while in the multiparae the labor time averaged about four and one-half hours.

In cases conducted along the lines of the technique described, I have encountered none of the distressing obstetric symptoms laid at the door of pituitary extract and "dry labor," nor have I had any cases in which the cord was prolapsed as a result of the procedure.

I believe that this method of inducing labor prematurely has a place in obstetric practice, and I can recommend it for your trial.

SEPTIC PHLEBITIS OF THE BROAD LIGAMENTS*

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DURING the past few years we have been called in consultation in several cases of what we diagnosed as septic phlebitis of the broad ligaments. All of these cases have been postpartum. In each instance it was interesting to note that the parturient history was one of difficulty of one sort or another during the labor. In some instances the delivery had necessitated the use of forceps, again the labor had lasted two or three days. In one instance a full term baby was dead when delivered. The interesting fact is that all of the cases gave the history of some difficulty in delivering the child.

SIGNS AND SYMPTOMS

These cases would go along apparently quite normal for from three to seven days when there would be a sudden rise of temperature, elevation of the pulse rate, increase in respirations (at times resembling that short, jerky respiration seen in pneumonia with a concomitant pleuritic involvement such as we see in empyema). The abdomen would be considerably distended with marked tympany. The patient would appear sicker than the temperature and pulse would warrant, in fact most of these patients seemed quite prostrated and acutely ill. They complained of marked tenderness over the outer regions of the lower abdomen with some dull and continuous pain in these regions. Most of them complained of quite a severe, occipital headache and of being extremely sick. They appeared very ill and seemed to have the expression of one septic and overcome by some sudden interruption to a normal postpartum convalescence. A number would lie recumbent with the thighs flexed upon the trunk and

the legs flexed upon the thighs as this posture seemed to give relief.

PHYSICAL EXAMINATION

These patients were extremely sick and prostrated, coming as this infection did supplementing the more or less severe task necessary even in a normal delivery. The features were generally of the anxious type, the face flushed and the pulse rapid and thready. The temperature was often elevated to 104°F. or 105°F. and there may or may not have been chills. These chills generally were fleeting but left the patient in full perspiration. The abdomen was tense even to the point of being rigid and board-like. For this reason the abdominal findings were not always clearly outlined. Percussion revealed only a moderate amount if any tympany. When tympany was present it was generally due to distention of the large bowel and principally the transverse colon, descending colon and sigmoid flexure. As the infection was located in the pelvis and not lymphatic in type very little paralysis of the alimentary tract was present.

A very moderate degree of digital pressure elicits markedly acute tenderness, extending outward along the broad ligament lines, in both lower quadrants of the abdomen. The delicate finger tips could detect a suggestion of induration or doughy sensation. In other words the deeper structures seemed to have lost their resiliency. With the thighs flexed on the trunk, the patients in recumbent posture, these sensations could be more readily brought out. By having the patient turn first to the left and then to the right it was noted that the areas of tenderness did not alter. Percussion elicited a moderate degree of dull-

* Submitted for publication March 2, 1929.

ness over the broad ligament areas. This sign could be best determined by very light percussion.

VAGINAL EXAMINATION

Vaginal examination—per speculum revealed the usual external lochea discharge from the patent os and from the pouty, edematous cervix. The vaginal mucosa was found somewhat abnormally redundant, edematous and at times tender to the irritation of the instrument. Culture of the lochea revealed the usual vaginal flora but seldom any extraneous organisms. Bimanual examination permitted one to outline very definitely the marked tenderness along the broad ligaments and that feeling of induration or of a doughy sensation. There was one sign which when found was considered most significant or even pathognomonic. This sign was elicited with the tip of the index and middle fingers of the examining hand. Gently these fingertips were placed against the lower end of the cervix and upon a somewhat sudden more or less gentle upward pressure against this part of the cervix the patient would complain of severe pain radiating outward along the course of the broad ligaments on both sides of the lower abdomen. If properly executed this sign was to be found constant and diagnostic in its significance. By careful notation one would notice that the fundus and cervix did not recede from the finger normally but quite slowly and that their return to full pressure against the finger tips occurred slowly. With these last two signs present, one could feel very sure that a phlebectasis of the broad ligaments was present. The lower extremities showed nothing of extreme importance. Edema was not often found and although the veins were occasionally somewhat distended this condition might be found in any postpartum patient.

The outstanding findings were tenderness along the course of the broad ligaments, a suggestion of induration or doughy feeling in these areas and the *digit tip sign* as found per vagina. These in themselves

carried much weight especially with the history, sudden onset, quite high temperature and possible chills.

LABORATORY FINDINGS

The urine was generally negative except that now and then a slight trace of albumin was found. The sediment, as a rule, did not reveal anything important. The specific gravity might be elevated a few points, while the kidney function was about normal. The blood revealed a leucocytosis of moderate degree (12,000 to 15,000). The differential count revealed a predominance of polymorphonuclear leucocytes. The coagulation time was often elevated two or three minutes. In other words the laboratory findings generally revealed a moderate degree of systemic resistance.

SURGICAL TREATMENT

We are dealing with very sick patients with the venous channels of the broad ligaments markedly tortuous and partially thrombosed in certain sections. If this partial thrombosis was found it revealed nature's endeavor to halt the progress of infection.

In our clinic the first two cases were not subjected to any surgical intervention and they gradually grew worse and finally succumbed to the infection, one four days and the other six days after the onset of the infection. The loss of these 2 cases led us to believe that possibly they could have been saved by multiple, intermittent ligation of the veins of postmortem findings. At the postmortem a moderate amount of cloudy fluid was found in the pelvic cavity. Careful dissection of the veins of the broad ligaments showed markedly thickened walls and here and there partial or incomplete thrombosis. This partial thrombosis was found in segments $\frac{1}{2}$ to 1 inch in length. This brought to our attention the fact that nature was making an attempt to stop the progress of the infection by endeavoring to occlude the venous channels. Thus we conceived the

idea of multiple, intermittent ligation of these veins. In subsequent cases we have practiced this method and all of the cases have survived. To some this might appear as rather radical in the face of an acute, highly potent infection but the results seem to warrant the procedure.

TECHNIQUE

A median, suprapubic incision is made and the intestines carefully walled off with gauze packs. The broad ligaments are then separately delivered and the largest, most tortuous veins are ligated with No. 1 chromic catgut. This ligation is done at various points in each broad ligament. No attempt is made to incise the veins between the ligatures. Any incising of the veins is avoided with the hope that simple, multiple, intermittent ligation will temporally shut off the circulation through these channels and after the acute condition is over and subsiding the ligature will be absorbed and the venous channels again become patent.

CONCLUSIONS

The exactness of this theory has been substantiated in 2 cases subsequently operated upon for other trouble. In both of these cases the broad ligaments were carefully surveyed and the venous channels found patent and normal in size and texture. This was a most convincing

sign to us that the procedure was well worth while. Again the fact that all cases, since the first 2, have recovered would seem to warrant surgical intervention. Of course, all of these cases might have recovered without any surgical procedure yet the fact that the first 2 cases coming under our observation were not ligated and died, and all of the subsequent cases were ligated and recovered would certainly warrant a continuation of the surgical procedure in subsequent cases.

Septic infection of the broad ligaments secondary to parturition or other intra-uterine conditions is a very serious condition. The fact that a general peritonitis was found in the 2 early cases that came to autopsy justifies the rather simple surgical procedure necessary to prevent extension. No form of drainage was used in any of the cases operated upon.

Following the operation all cases are placed in the modified Fowler's position and hot boric-acid fomentations, changed under strict aseptic precautions, every two hours, applied to the abdomen. It is an interesting fact that these last two procedures were also instituted in the first 2 fatal cases. We feel that these procedures, by fortifying the abdominal wall, aid much in the recovery. Sterile-water vaginal douches, temperature 110°, are begun on the second day and continued as long as the temperature remains the least bit elevated.



THE SYMPTOMS & DIAGNOSIS OF URINARY STONE*

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THE diagnosis of urinary stone depends almost wholly today upon roentgenography. This applies more especially to stones located in a kidney or a ureter; but likewise, stones in the bladder are for the most part easily recognized by roentgenography. In the detection of stones in the bladder, however, roentgenography has to divide honors with the cystoscope and the stone searcher. So important a part does roentgenography play in the diagnosis of cases of urinary lithiasis that our first problem in any patient suspected of stone is to secure good roentgenograms. We have frequently to carry out certain cystoscopic maneuvers in conjunction with roentgenography because of certain limitations that we have found it possesses in the study of cases of urinary stone. These supplementary maneuvers are for the most part the passage of roentgen-ray catheter with or without the filling of a kidney pelvis or an ureter with a shadow-casting fluid for the purpose of making clear the relation of a given shadow to some part of the urinary tract. This combination of a good roentgen-ray with the use of a shadow-casting catheter, a pyelogram or a pyeloureterogram will allow us to draw very accurate conclusions in all but a few most puzzling instances of urinary stone.

If, then, urinary stones are, with some exceptions which we shall consider later, so easily recognized by roentgenography, why is it that we see patients whose symptoms have persisted for years, but who have never been roentgenographed though occasionally they have been subjected to various surgical procedures without relief? Most often these operations were for appendicitis; less frequently for gall

stones, adhesions, or some pelvic condition; procedures to which they presumably would not have been subjected had they been roentgenographed. This is due, in my opinion, to the fact that it is hard to do away with old beliefs and traditions; and for some generations before the introduction of roentgenography only such stones in the upper urinary tract were recognized as were attended with great pain or with hematuria, or both. The actual pain of urinary stone, however, often falls so far short of the traditional requirements set for it that the condition is often not even suspected and roentgenograms are therefore not made. Formerly when a renal stone that had not caused much pain was found at autopsy or otherwise, it was spoken of as a "silent stone," and the inference was that this was a very unusual condition. More recent experience has taught us that this is not so; that excruciating pain is, to be sure, a common symptom in such stones as offer an obstruction to the outflow of urine, either at the kidney outlet or in a ureter, but that a stone which is fixed either in a calyx or in the pelvis of a kidney not only need not, but as a rule does not, give excruciating pain.

It has been my experience that only the blocking of the urine in such a way as to overdistend the kidney capsule quickly gives the pain that is called renal colic, commonly considered the necessary accompaniment of a kidney stone. In several instances I have been able to stop pain of this type literally in a minute or two by passing an ureter catheter past an obstructing stone and drawing off the accumulated urine. In one such case that I saw some years ago I drew off between 50

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and 60 c.c., giving immediate relief to great pain; the next day when I did a pyelogram on the same kidney it would hold only between 6 and 8 c.c., showing the grade of acute hydronephrosis that had been present. While one may say that this very acute type of pain, due to overdistention of a kidney pelvis, is usually due to an obstructing renal stone, one may not say that all or that even most renal stones produce it.

It is much the same with pain extending down into the testis in the male; when present it is quite characteristic, and usually indicates a stone low in the ureter of the corresponding side. It is not present, however, in a large proportion of cases.

The pain that we do meet most often, in patients with stones in the kidney that are not producing obstruction to the outflow of urine, is a dull ache in the back or in a loin, or a dull stomach ache; a grade of pain that may be produced by many conditions other than renal or ureteral stone. That this pain is not of necessity very acute nor very characteristic is attested by the fact that one sees patients who have borne it for a very long time. In a patient whom I saw some years ago this pain had been present for thirty-five years. In some patients the dull pain of stone in a kidney is so high in the abdomen that it may be considered due to an ulcer of the stomach, and the patient may be treated for this trouble for a long time. Per contra, at least 2 patients have been sent to me for pain in a loin, supposed to be due to renal stone, which pain on careful study proved to be that of a peptic ulcer. A predominance of gastric symptoms appeared in 15 per cent of a small series of cases of renal stone that I went over a few years ago, though these patients all showed some pain in a loin or in their back, as well as their gastric symptoms.

In a case seen a week or so ago, a man with a stone that caused an intermittent blocking of his left kidney pelvis suffered only from attacks of chills, fever and a turbid urine. He had never had any pain

in spite of the fact that he had not only a stone in his left kidney but a big gall stone as well. It is evident that the pain shown by a considerable proportion of patients with renal and ureteral stone is not the characteristic pain of renal colic.

Hematuria has long been considered a very characteristic symptom of stone in the upper urinary tract, yet in a series of cases of renal stone that I studied only 30 per cent of the patients had a history of having shown a urine that was bloody to the eye. Even microscopic blood may be absent in the urine of a patient suffering from a renal stone, in the specimen or specimens that one happens to examine, though it is probably present at some time in the urine of every patient who has an urinary stone. In my experience kidney bleeding that recurs after the trauma of exercise is very suggestive of a renal stone; but only suggestive.

Moderate tenderness is usually an accompaniment of renal colic and ordinarily subsides with the acute attack. A mass in a loin is met only in the stone patients in whom there is a calculous pyelonephritis or calculous pyonephrosis. The condition is usually accompanied by a large amount of perirenal thickening. The patient may have chills and fever. This indicates a pretty well destroyed kidney.

The presence of pus in the urine accompanied by more or less bladder irritation may, in a few instances, be the chief objective and subjective signs that a urinary stone has produced even a stone in a kidney. Bladder irritation alone may be the outstanding symptom in an occasional case in which a stone is caught low in a ureter.

Nausea and vomiting are common accompaniments of a renal or ureteral stone that is causing renal colic, that painful condition that results from overdistention of the renal capsule, whether that distention is due to an obstructing stone, an acute hydronephrosis, or an overfilling of the renal pelvis in making a pyelogram. It presents nothing that is

characteristic in the case of stone. The nausea and vomiting that has its origin in a renal colic is often prolonged by the injudicious use of morphia.

It will be seen that if we are to recognize stones of renal origin early, whether they are in a kidney or an ureter, at a time before they have done irreparable harm, we must not wait until we have classical symptoms before we submit the patient to roentgenographic study as to pathognomonic symptoms of this condition.

In this connection I want to point out a danger that I have seen arise from putting too much weight on a single sign. In two instances I have seen patients with severe right-sided pain who were under the care of most conscientious, careful men, in whom operation had been delayed because what were supposed to be red blood cells had been found in the urine. Both these were cases of appendicitis rather than stones in the ureter, and operation when done proved to be too late. The urines in both cases showed some crystals of sodium urates, which were of a size to simulate very closely red blood corpuscles. In these instances an attempt to be most fair to the patients had led to the placing of too much stress on one sign, with disastrous results. The diagnosis in these patients must be on the weight of evidence, not on a pathognomonic symptom or sign.

To summarize regarding the symptoms of urinary stone I can do no better than to quote from a previous paper: "Pain, varying in intensity and location, is the most common symptom shown by renal stones; therefore, even vague pain that persists and is not accounted for satisfactorily, whether this pain is in the loin, back, or upper part of the abdomen, should bring to one's mind the possibility of a more or less 'silent' renal stone; especially is this true when this pain is accompanied by a hematuria or even the presence of microscopic blood in the urine. A gross hematuria that appears quite regularly after exercise, such as riding or driving, is very suggestive of stone.

Patients suffering from so-called 'dyspepsia,' for which no adequate cause is found should be investigated as to the possibility of a renal stone, as should all cases of renal pyuria of undetermined origin." These symptoms should lead us to the careful roentgen ray study of any patient presenting them.

Early in this paper I stated that in our detection of stones in the urinary tract we have to rely chiefly upon good roentgenograms. While a roentgenographer might not agree with me as to what constituted a "good roentgenogram" of the urinary tract, I have very definite ideas of what gives me the most help. If there is any question of the patient having a barium meal or a barium enema, as is often the case, the plates of the urinary tract should be taken first and the whole roentgenographic investigation of this tract carried out before that of the digestive tract is begun. Large masses of barium in the intestines obscure the urinary tract and small collections give shadows that may be confusing.

The first requisite of a good roentgenogram is that the whole stone-containing area shall be taken in the first set of plates, which should include plates of both renal regions and both ureters, as well as the bladder. These plates will show less distortion if one plate is made of the renal region and the upper course of the ureters, and then another overlapping plate of the lower part of the ureters and bladder. This taking of the whole urinary tract is exceedingly essential. I have known two instances where a kidney has been removed because the original roentgenogram included only the painful renal region while the obstruction that caused the trouble was low in the ureter but was not discovered until after a nephrectomy had been done. While succeeding roentgenograms may with advantage often be confined to a limited area of the urinary tract, the first should always include the whole tract.

Another requirement for a satisfactory

renal roentgenogram is that it shall show the outline of the kidney, and that this area shall not be obscured by gas shadows. If in a roentgenogram that measures up to the above requirements we see no shadow, we can rule out pretty safely all but a few uric acid stones, and for the most part these will be so small as to be considered "non-surgical;" that is, one probably would not submit them to operation if he had found them.

The density of a pure uric acid stone is so near the density of water that it shows little contrast when in water. I have been quite unable to get a roentgen-ray shadow of small uric acid bladder stones that I could see plainly with a cystoscope. It seems possible that a uric acid stone, even of some size, might give no shadow if it were in a moderately dilated and distended kidney pelvis. I have no knowledge of having overlooked one of any size, though I have overlooked a number of small uric acid, renal or ureteral stones that were passed later; so many, in fact, that I always warn a patient whose symptoms are very suggestive, but in whom I can demonstrate no shadow, that it is perfectly possible he may later pass a small stone which I have been unable to see, but that had I seen it I probably should not have advised operation.

Some of the shadows that we find in the kidney region in cases of suspected renal stones we can put down at once as positive. If we find a coral-like shadow overlying a kidney outline we may say without fear of error that the patient has a renal stone; in some other shadows of the triangular, dumb-bell or peach-stone type we may feel pretty sure of the diagnosis of renal stone.

Besides the above mentioned shadows there are a good number that are not definite. Some of these are of material in the intestine. For the most part these will be irregular in shape. They may be hardened feces, bits of foreign material like pieces of bone, or little areas of barium in case the patient has had previous gastro-

intestinal roentgenograms. These shadows may overlie the kidney area in the first roentgen-ray plate, but will almost invariably change their position in succeeding plates. Often their relation to the inlying ureter catheter or to the pyelogram will be such as to show at once that they are not connected with the kidney. Gas shadows are always dark and cannot be confused with the lighter shadows of stone. The way in which they annoy is by overlying and obscuring the stone-bearing area of a kidney.

The pain of cholelithiasis may occasionally be confused with that of stone in the right kidney, and the shadow of a gallstone for that of renal stone. If the kidney outline is present it will often be found that the gallstone shadow lies to the right of this, which in itself will serve to differentiate the two. I regret to say, however, that though the kidney outline is a requirement for a satisfactory renal radiograph one does not always get it. The larger gallstones will often show a concentric structure that we rarely see in renal stones; others will appear as shadowy rings. Certain gall bladders filled with small stones may show an accumulation of shadows consisting of little rings. These will not simulate renal stones sufficiently to offer any real difficulty to the person who has seen any considerable number of them. In the few cases in which a gallstone has been of such shape and density as really to resemble a kidney stone, the pyelogram has been sufficient to distinguish between the two.

Very rarely calcified tuberculous areas on the surface of a kidney, perfectly round in shape and about 3 or 4 mm. in diameter, may so overlie the stone-bearing area of a kidney as to simulate very closely a stone in the kidney. I have seen 2 such cases. One, seen a number of years ago, I had the opportunity to study but failed to make a correct diagnosis. I cut down upon this kidney, found the calcified area that corresponded with the roentgenograph and curetted it. There was an immediate dis-

semination of the tuberculous process through the whole kidney, which I had to remove shortly after. This man finally made a good recovery but was ill for a year or more. The other case, which I saw in consultation, had to have a nephrectomy following curetting of the calcified area, but died shortly after of a generalized tuberculosis. I know of no way of making a definite differential diagnosis in this condition. If one should, however, cut down on such a cortical area, my experience has been that it is an extremely wise thing to let it alone.

A wart or pigmented mole on the back may give a shadow that is confusing when it overlies a kidney outline. A case of this sort has been reported by Alcock,¹ and I have had the opportunity of seeing two instances where the shadow of such a mole or wart on the back gave the roentgenographic appearance of a urinary stone. In all these instances the true nature was recognized and no operation was attempted other than the removal of the wart, after which the shadow disappeared.

A calcified abdominal gland is the condition that most frequently simulates a urinary stone. These calcifications, so far as I can determine, occur in both the mesenteric and the retroperitoneal glands. It is the supposed end-result of a tuberculous invasion of the gland, although I do not know that it is always a tuberculous process. These patients present vague abdominal pain that suggests the possibility of urinary stone. The calcifications, which it is supposed are not in the glands that produce the pain but in other glands in which nature has already healed the inflammatory process, may give shadows that are very difficult to distinguish from the shadow of a renal or ureteral calculus. As a whole these shadows have a mottled appearance that is rather suggestive. Another point which often serves as a differential sign is the fact that a calcified mesenteric gland is very apt to show a

change in position in two roentgenograms. This when combined with the mottled appearance will serve to make the nature of the shadow clear in many instances. The calcified retroperitoneal gland is fixed, but it has the same mottled appearance as that of the mesenteric gland. The differentiation of these conditions will often require the use of the roentgenographic catheter and the pyelogram. Usually these will serve to distinguish them.

The symptomatology of ureteral stones is much the same as that of renal stones, with the exception that a larger proportion of them present renal colic, the obstructive pain formerly considered typical of all kidney stones. Their roentgenographic diagnosis is much simpler than that of renal stone, especially when they are situated in the mid-portion of the ureter. There are few ureteral stones, however, that I care to cut down upon without verifying my diagnosis by the passage of a roentgen-ray catheter or by the making of an ureterogram. The general course of the ureter is a common place in which to find the shadows of calcified mesenteric glands; and the passage of a roentgen-ray catheter will usually show so conclusively whether a suspected shadow is in the ureter or not that I rarely fail to use it. If there is any question, one can take stereoscopic plates, or an "offset plate" (one in which two exposures are made on the same plate, the second after moving the roentgenographic lamp). If in a plate of this sort the questionable shadow bears the same relation to the roentgenographic catheter in both exposures, it is practically conclusive evidence that the shadow is in the ureter. Sometimes a more conclusive test is to fill the ureter with shadow-casting fluid and withdraw the catheter. Occasionally the column of fluid is held above the stone very definitely; at other times the stone will stand out as a definite change in density in the column of fluid. The methods of differentiation will have to be used most frequently in the case of questionable shadows in the lower part of the ureter,

¹ Alcock, N. G. A pigmented mole casting the shadow of a renal calculus. *Am. J. Roentgenol.*, 5: 277, 1918.

the portion within the anatomical pelvis. Here we have not alone an occasional calcified gland but the shadows of calcified pelvic vessels, as well as numerous phleboliths, to differentiate from the ureteral calculus. In spite of these difficulties I believe the careful study of any questionable shadow in the lower end of the ureter, by noting its relation to a roentgenographic catheter both in the stereoscopic picture and the "offset" plate, together with its behavior with the filled ureter, will allow one to draw an accurate conclusion in almost all instances. This last precaution, had I known it at the time, would have kept me from doing two unnecessary operations that I carried out a considerable number of years ago. In these instances I operated upon shadows that I supposed were small stones low in the ureter but which proved to be phleboliths either in a vein of the ureter or in a vein that was closely adherent to it. These stones were extremely small and fairly well down in the ureter. They were stones which I would not think of subjecting to operation today, even if I knew they were in the ureter, as they would certainly be passed.

There is one precaution that I wish to mention in regard to ureter stones and that is, if one plans to remove a stone from an ureter which the ureterogram has shown is dilated, even if not very much dilated, it is extremely wise to have a roentgenogram taken immediately before operation. A few months ago the taking of such a roentgenogram immediately before operation showed me that a stone which had given rather severe symptoms over a long period of time and which my original roentgenogram had shown was well up towards the kidney, and in a somewhat dilated ureter, was on the morning of operation down very near the lower end of the ureter. Several times the observance of this rule has saved me the embarrassment of having to explore another part of the ureter in order to remove an ureteral stone.

The symptoms of bladder stone may be described briefly as those of bladder irritation or urinary distress. When we come to the diagnosis of bladder stone, which may be said to be relatively simple, roentgenography, while it occupies a useful and important place, does not occupy the absolutely pre-eminent position that it holds in the diagnosis of renal and ureteral stones. It has to share its place with the cystoscope. Most bladder stones may be detected by either roentgenography or cystoscopy. Both, however, have certain limitations. A judicious combination of the advantages of both makes diagnosis easy and sure.

We will first consider the limitations of roentgenography; and curiously enough one of them is that it will occasionally show too much. I refer to a few women suffering from bladder discomfort who have on roentgen-ray examination, showed what looked to be a big stone in the bladder, but which was really a calcified fibroid in the anterior wall of the uterus. Cystoscopy showed the bladder to be free of stone. The condition is a relatively rare one.

It has been my experience that pure uric acid stones, as well as the xanthine stones, will not be shown by roentgen-rays, especially if the pictures are taken with a lot of fluid in the bladder. Of course these will be shown by the cystoscope, and may be felt with the stone searcher. The use of the cystoscope is the only way I know to determine the nature and position of a stone that has formed on a ligature and hangs down from the vault of the bladder. While I do not doubt the roentgen ray would have shown the stone in the cases I have seen, I do not believe it would have shown features that it was very necessary to have in order to deal with the condition properly, and which were brought out clearly by cystoscopy. The above are certain limitations of roentgenography as applied to the diagnosis of bladder stones.

On the other hand, two types of bladder stone that I have seen will be clearly made

out only by a combination of roentgenography and cystoscopy. One is the rare condition in which a stone is entirely concealed in a diverticulum of the bladder, and its nature only made evident when an ureter catheter is passed through the diverticulum opening and is found on roentgenography to be touching the stone, or when one makes a cystogram. The same thing applies in the shirt-stud type of diverticular stone, a condition in which the roentgen ray will show a dumb-bell or shirt-stud shaped stone while cystoscopy will make clear that only one part of it lies in the bladder. These differentiating points are essential when it comes to the question of operation.

I have seen the lack of them lead to confusion and incomplete operation.

One may summarize by saying that the symptoms of urinary lithiasis may be any degree of lumbar or abdominal pain, accompanied or not by evidence of some abnormality of the urinary tract. The diagnosis of lithiasis will in most instances be made by a roentgenographic study of the patient, supplemented in many cases by the addition of certain cystoscopic maneuvers which are necessary because of the limitations that unaided roentgenography offers in differentiating between the various shadows that appear in the abdomen and pelvis.



CHONDRO-OSTEO-DYSTROPHY*

ROENTGENOGRAPHIC & CLINICAL FEATURES OF A CHILD WITH DISLOCATION OF VERTEBRAE

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THE patient, a boy (W.T.B.) aged three years nine months, was a full term intelligent little fellow and looks relatively well nourished, but the mother says that he has lost 1 pound during the past three months.

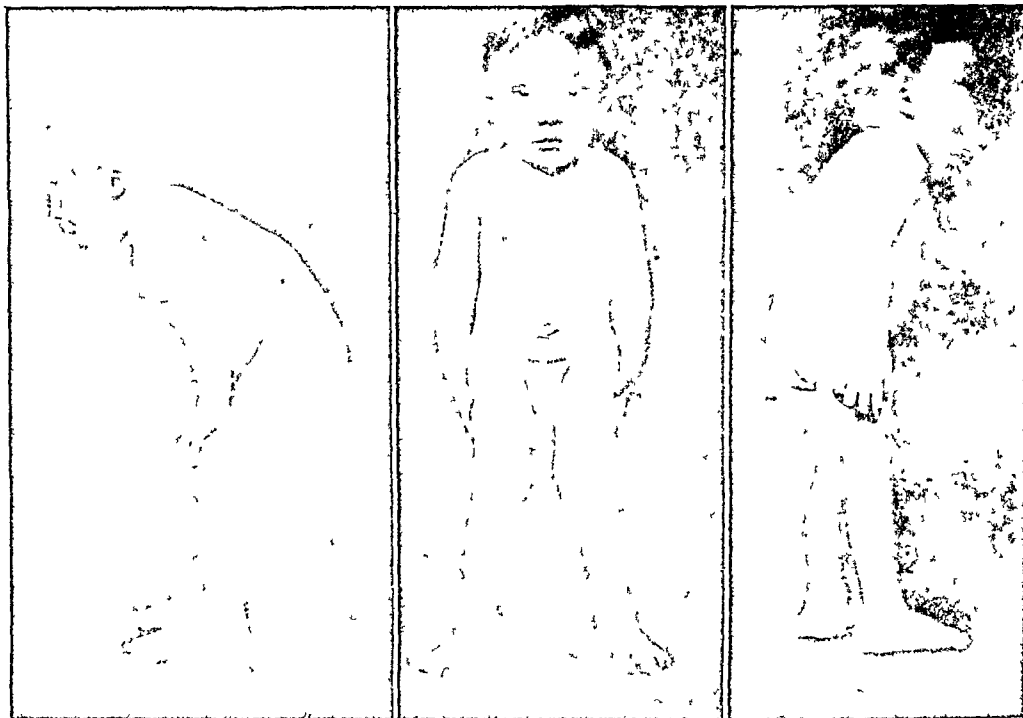


FIG. 1.

FIG. 2.

FIG. 3.

FIG. 1. W. T. B., aged three years nine months, assuming position in which he first stood and walked. Note general kyphosis with prominence of lumbodorsal junction, the site of dislocation, also degree of swelling in wrist, knee and ankle joints.

FIG. 2. W. T. B. Note large joints and double inguinal hernia.

FIG. 3. Note short thick neck and large wrist and prominence of lumbodorsal spine.

he began to walk at thirteen months of age. The mother then noticed that the child could not stand erect but stood and walked with the body supported by the hands on the knees as in Figure 1.

On this account he was taken to the hospital but no definite abnormality was detected. Some months later he was examined at another institution and was provided with a posterior spinal support. Similar appliances have been worn continuously since.

Present Condition. The boy is a bright

He is 2 feet 9 inches in height and weighs 26 pounds 11 ounces. He can stand almost erect without supports and walks with a normal gait but readily gets tired and then lies down and assumes the hand on knee position during his sleep. It will be seen from Figures 2 and 3 that his joints are all on the large size; there is no evidence of wasting. The neck is short and thick and he has a double inguinal hernia.

All his teeth show marked caries.

He had pneumonia when twenty months old but since has had no other illness. He has a

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good appetite and for nearly three years has been given daily doses of cod liver oil. The parents, who are normal in stature and appearance, have had one other child (a girl) previously, which appeared to be quite normal but died of pneumonia at the age of twenty-one months. No history of any deformity in any member of the parents' families could be obtained from them.

The Roentgenographic Appearances of the Skeleton. The most noticeable features are the large joint spaces; the irregularity and fragmentation of the epiphyses particularly of the metacarpals and metatarsals; the irregular shape and size of the vertebral bodies and the dislocation of the lumbodorsal vertebrae. The short, thick, long bones and the coarse, irregular reticulation of the cancellous tissue and the absence of the regular lines of the lamellae.

For the purpose of comparison I have charted the features of individual bones in this case with a description taken from an average of a number of boys at this age period (Table 1).

TABLE I
COMPARATIVE DESCRIPTION OF ROENTGENOGRAMS

Area	Normal	Chondro-osteo-dystrophy
Spine (Anteroposterior roentgenograms) (Fig 4)	Bodies of vertebrae oblong in shape with slight convexity of superior and inferior surfaces Uniform in density, fine reticulation of cancellous tissue Transverse processes uniform in density, regular in outline, convex lateral extremities Articulations arranged in two straight lines which gradually diverge from upper dorsal to upper sacral vertebrae Intervertebral spaces regular, gradually increasing from upper dorsal to lower lumbar	Bodies irregular in outline and shape Outlines difficult to define particularly in cervical, upper three dorsal and all lumbar vertebrae Structure indefinite and mushy Transverse processes irregular in structure and outline with concave lateral extremities Articulations irregular in alignment and spacing Intervertebral spaces irregular and ill-defined
Spine (Lateral roentgenograms) (Fig 5)	Regular in outline and density, rectangular in shape with slightly concave anterior borders sometimes with median notch or groove extending towards the posterior surface Spinous processes regular in outline and density with convex posterior extremities	Cervical bodies irregular in outline and density Dorsal bodies, upper three irregular in outline and "mushy" in appearance The lower dorsal bodies have "tongued" anterior surfaces and are irregular in size The lumbar bodies are irregular in size and shape There is a forward dislocation of the dorsal vertebrae on the 1st lumbar, the anterior surface of the

TABLE 1 (Continued)

Area	Normal	Chondro-osteo-dystrophy
Skull (Fig 6)		body of the 1st lumbar being on a plane with the posterior border of the 12th dorsal The articular processes are small and irregular Spinous processes short and stunted Marked caries of all the teeth Sella turcica normal in size and shape No definite departure from the normal bone structure or shape
Ribs (Fig 5)		Thicker than normal with expanded heads and anterior extremities Three "spotted" rounded centers of ossification of epiphysis Upper end of diaphysis thickened and irregular in shape Irregular in outline Considerably wider than normal
Humerus (Upper end) (Fig 7)	Regular in outline epiphysis assuming characteristic features	Thicker and shorter than normal Compact tissue appears to be thinner than normal Fragmented appearance of external condyle
Shoulder joint (Glenoid)	Regular joint space	Thicker (particularly upper thirds) and shorter than normal with irregularity of superior and inferior extremities Radial epiphyses irregular
Humerus shaft		Os magnum, unciform beginning to assume characteristic features Outline smooth and regular
Humerus (Lower end) (Fig 8)	Regular and uniform in structure	Proximal extremities rounded with tendency to assume characteristic features
Ulna and Radius		Proximal extremities rounded with tendency to assume characteristic features
Carpus (Fig 9)	Os magnum and unciform beginning to assume characteristic features Outline smooth and regular	Proximal extremities rounded with tendency to assume characteristic features
Carpal space		Proximal extremities rounded with tendency to assume characteristic features
Metacarpals		Proximal extremities rounded with tendency to assume characteristic features
Phalanges	Distal extremities regular convexity	Proximal extremities rounded with tendency to assume characteristic features
Pelvis (Fig 10)	Inner margin of pelvis and symphysis pubis "champagne glass" shaped being about 3 1/4 inches wide at the top and 1 1/4 inches deep	Inner margin of pelvis and symphysis pubis "wine glass" shaped being about 2 1/4 inches wide at the top and 2 inches deep
Joint Spaces		Inner margin of pelvis and symphysis pubis "wine glass" shaped being about 2 1/4 inches wide at the top and 2 inches deep
Hip	1/4 inch between "articular" surfaces	1/4 inch between "articular" surfaces
Sacroiliac	1/4 inch between lateral border of sacrum and medial border of ilium	1/4 inch between lateral border of sacrum and medial border of ilium
Symphysis	1/4 inch between bony points	1/4 inch between bony points
Bone Structure	Fine reticulation, borders regular and well defined	Coarse reticulation, borders irregular Less radio-opaque Relat-

TABLE 1 (Continued)

Area	Normal	Chondro-osteo-dystrophy
Sacrum	Regular. Joint lines well defined.	tive sclerosis of joint boundaries. Irregular, joint lines and spaces irregular and wider.
Femur (Head Epiphysis)	Regular in outline and density. Joint surface: regular convexity.	Small, irregular and fragmented with marked irregularity of diaphyseal extremity.
Femur Shaft		Shorter and thicker than normal.
Lower Extremity (Fig. 11)	Uniform in density and regular in outline.	Small areas of increased density particularly inner condyle. Periphery denser.
Knee Joint Space	$\frac{3}{16}$ inch midline.	$\frac{5}{16}$ inch midline.
Tibia (Upper End)	Regular outline; uniform in density.	Periphery denser. Irregular outline. Not uniform in density.
Tarsus (Fig. 12)	Regular in outline assuming characteristic shape, uniform in density. Lamellae striation clearly visible and regular.	All present but irregular in outline and shape. Definite spur on plantar surface of os calcis. Sclerosis of periphery of superior and inferior surfaces of astragalus and os calcis. Coarse reticulation of cancellous tissue. No regular disposition of lamellae.
Metatarsals (Fig. 13)	Rounded regular extremities with tendency of proximal ends of 1, 2, 3 to assume characteristic shape.	Proximal extremities irregular and conical in shape. Coarse reticulation of cancellous tissue. Epiphysis of 1st metatarsal irregular and fragmented. Epiphyses of other metatarsals very irregular and "fringed" with denser points of ossification. Distal extremity of 1st metatarsal very irregular.
Phalanges		Shorter and thicker than normal, no other definite change.

The features shown in the roentgenology of this patient are rarely seen. Isolated joints or areas of the skeleton are seen which present some of the features.

Achondroplasia. Irregularities of the carpus, tarsus, metatarsals and metacarpals as well as the long bones are seen in the roentgenograms of achondroplastic dwarfs. These changes according to Siegert and others may be found on one side of the skeleton only. Siegert shows a roentgenogram of the hand of a thirteen-year-old chondrodystrophic dwarf with somewhat similar changes.

Silverskiöld reported an abnormal case of chondrodystrophy under the title "*A forme fruste*" of chondrodystrophia with changes simulating several of the known

local malacias. The roentgenograms of his patient show vertebrae of similar shape to this case, also large irregular acetabuli, marked deformities of the femoral epiphyses and necks of the femora, irregularity of outline of os calcis and astragalus. He says:

Here we have a peculiar case with several osseous changes, especially the extremities, most pronounced in the lower ones. There are marked skeletal anomalies, such as epiphyseal formations in foot and hand which normally do not exist (first metatarsal, first and second metacarpals, basal phalanges of thumb and great toe) as well as free osseous nuclei in carpus, tarsus and other locations.

He contrasts this case with Legg-Calve-Perthes-Waldenström's change in the hip joint, Sven Johansson-Sinding-Larson's change in the patella apex, Osgood-Schlätter's change in the tuberosity of the tibia, Haglund's change in the calcaneal apophysis, Köhler's change in the scaphoid of the foot and heads of the second and third metatarsals, Kienbock's change in the semilunar, Kappis' changes in the apophyseal nucleus of the internal epicondyle of the humerus and capitulum humeri and Möller's changes in the skeleton of a cretinoid dwarf.

Local Malacias. These are for the most part enumerated in the previous paragraph. The roentgenograms of the bones in these cases show changes which bear little resemblance to those of this case. The changes shown in the roentgenograms of the vertebrae of patients with osteochondritis of the spine (Buchman), kyphosis dorsalis juvenilis of Scheurmann, are also dissimilar.

Cretinism. The roentgenograms of the epiphyses of some cretins, as in the case reported by Möller, show changes which resemble those shown in this case. This diagnosis had never been considered in this patient, consequently thyroid had not been tried. The effects of a course of thyroid will be observed.

Syphilis. The roentgenographic features of this case do not suggest syphilitic changes. The Wassermann reaction was



FIG. 4

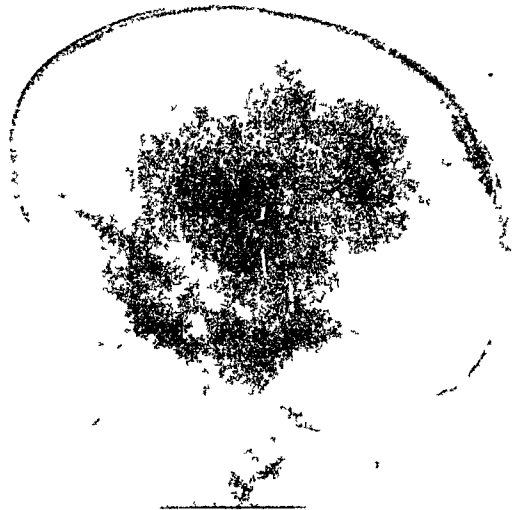


FIG. 6



FIG. 5.



FIG. 7.



FIG. 8.



FIG. 10.



FIG. 9

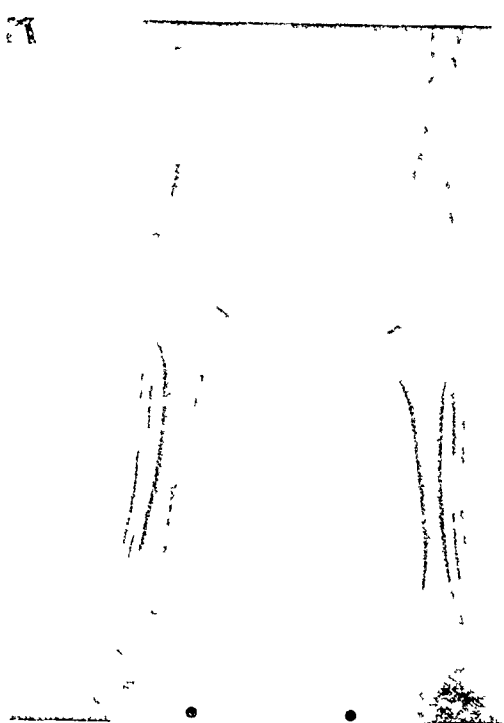


FIG. 11

negative and no evidence of syphilis of the parents could be obtained.

Other Conditions. Hanson shows the

which has produced a disturbance in the normal growth of cartilage and bone. During the last three to six months the



FIG. 12.

roentgenogram of the spine of a child of six with similarly shaped lumbar vertebrae but he states that the roentgenogram was found in the collection of H. Waldenström and that he could not trace the patient or any records. He gives the details of two other patients in the same series but the patients are aged thirteen and fourteen years and the roentgenographic appearance does not resemble the first case.

Wollenberg illustrated a congenital forward dislocation of the vertebra on the 2nd lumbar which is smaller than the other bodies. There can be no question that in this case there is some systemic affection

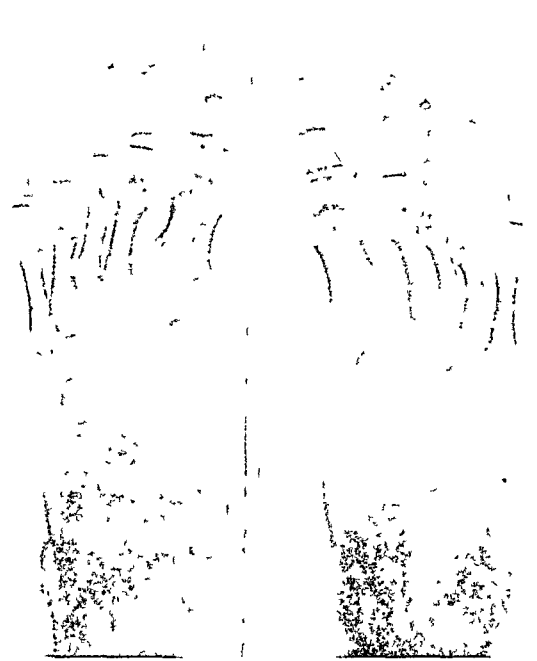


FIG. 13.

child has gradually lost weight and his condition has declined.

In conclusion I wish to thank Mr. Naughton Dunn for his permission to investigate and record this case, Sister Manaton and Nurse Thomas for their help with the illustrations, my secretary Miss E. Stewart for her assistance with the preparation of the manuscript, also Mr. Powell, Librarian of the Royal Society of Medicine, for his help with the literature.

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RADIOHUMERAL BURSITIS (TENNIS ELBOW)*

REPORT OF 2 CASES

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AMONG the disabilities of the elbow joint is one which has been at various times described as epicondylitis, epicondylagia, tennis elbow, epicondylus neuralgia, and radiohumeral bursitis. The affection is characterized by pain in the neighborhood of the external epicondyle of the humerus following some strenuous exercise, and by marked pressure tenderness in that area. The clinical symptoms and signs are remarkably constant, yet there is considerable difference of opinion in regard to the etiology, pathology and treatment of this condition. Although it is self-limited in its course, it is very resistant to conservative treatment.

The relative scarcity of literature on this subject indicates that the affection is not very common. It was first mentioned in 1873 by Runge, who obtained a cure in 1 case of two years' duration, by deep cauterization of the skin over the area of tenderness. Bahr in 1900 reported 3 cases, 2 of these being in tennis players. Still later there appeared articles by Clado, Preiser, Franke, Bernhardt, Vulliet and others. Osgood in 1922 first described inflammatory changes in a bursa, situated underneath the conjoined tendon of the extensors, anterolateral to the radiohumeral joint. He also demonstrated that removal of this bursa, when involved, produces a complete and permanent cure.

ETIOLOGY

Incidence. Fischer in 1923, collected 82 cases in the literature. Of these 49 were in men and 33 in women. The age of greatest susceptibility was forty to fifty years. The right elbow was involved more often than the left. Only 2 cases were bilateral.

Trauma. Direct or indirect trauma is mentioned as a prominent factor in the production of this condition in most cases. While the term "tennis elbow" suggests lawn-tennis as a frequent cause, it is often seen as a result of fencing, boxing, striking with a hammer, or other activities.

The following theories have been advanced in regard to the etiology of this disability on the basis of trauma; injury to the periosteum (Blecher, Momburg), tearing muscular attachments (Coues, Clado) injury to the radiohumeral capsule (Bahr, Dubs), radial incongruence (Preiser), traumatic neuritis (Winkworth, Marshall).

Toxemia. Several authors have explained this condition on the basis of toxemia. Franke's cases all followed influenza and trauma was never considered as a cause. Fischer maintains that it is dependent on primary toxic causes, or is produced by extension of inflammation due to injury of adjacent structures.

Osgood states that trauma is usually the principal factor but he has also seen a similar clinical picture, associated with lead-poisoning. On the other hand, Schlatter was unable to name any definite cause.

PATHOLOGY

This important phase of the subject has received very little attention, due to the fact that in most instances conservative treatment was employed and material was not available for study. Franke found, in 1 case, gross inflammatory changes in the epicondyle, but reports no microscopic findings.

Goedel, after removing the epicondyle in 2 cases, reports the "bone surrounded

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by scar tissue, with metaplastic bone formation" in 1 case; in the other case he found "partly calcified cartilage and periosteal new bone formation."

Bergmann has failed to find any evidence of inflammation in the muscles of the conjoined tendon.

A cystic degeneration and calcification of a subcutaneous bursa over the epicondyle have been described by Schmitt.

Osgood reports chronic inflammatory changes in the radiohumeral bursa, removed at operation. The presence of a bursa in the neighborhood of the epicondyle, as described by Osgood, has been disputed by Fischer, but it is apparent that he misunderstood the description in regard to the location.

SYMPTOMATOLOGY AND ROENTGENOGRAPHIC FINDINGS

The clinical symptoms of this disability are very typical. Following some strenuous use of the arm, there is a feeling of discomfort, or at times, acute pain in the region of the lateral epicondyle. This may be the result of a single trauma or may be due to oft-repeated trauma. The pain usually subsides temporarily, but returns when use of the arm is resumed. There is a very definite sense of weakness of the forearm, especially noticeable when the patient tries to hold or grasp articles with his fingers. Radiation of pain down the forearm is common. Such tasks as writing or winding a watch are difficult and painful.

Examination elicits a point of extreme tenderness in the region of the lateral epicondyle. This is very definitely limited to an area, several centimeters in diameter, over the radiohumeral joint, and somewhat anterior to it. Usually nothing else is found on palpation. The patient, however, complains of pain in that area when forcible extending the wrist or fingers or closing the hand tightly. Supination, especially when resisted, and full pronation are likewise painful. Very little or no discomfort is produced by passive motion.

Although the radiographic examination usually shows nothing abnormal, several different authors report pathological changes. Blecher and Bergmann have found a periostitis of the lateral epicondyle. Preiser established his theory of "radial incongruence" on roentgen-ray appearances. According to this author, the contour of a normal epicondyle is on the same plane as the head of the radius, whereas, in case of radial incongruence, the head of the radius projects beyond the epicondyle. This constitutes a static disturbance and predisposes to "idiopathic" arthritis deformans. The elbow with static disturbance also shows a more or less high-grade cubitus valgus.

The symptoms and objective findings make it apparent that the lesion is in close proximity to the conjoined extensor tendon, as they are aggravated by the movement of any of the muscles comprising this structure.

Neurological symptoms, in the form of paresthesias or even anesthesia, have been found in the areas of distribution of some of the cutaneous nerves in the forearm. These have led, in some instances, to a diagnosis of neuritis, traumatic or toxic. The radiating pain, so common as a complaint, is probably a referred pain, similar to that found in the thigh and knee, in certain diseases of the hip joint.

The bony relations mentioned by Preiser, when present, may well be a source of mechanical irritation to the soft tissues adjacent to the joint; however, they are too inconstant to be considered in every case.

COURSE AND PROGNOSIS

The duration of the disease is variable. Ordinary treatment has very little beneficial effect, and in the course of six months to three years, the symptoms usually subside completely even without treatment. Recurrences are common. An unusual case is that of Ochsenius who reported remissions and recurrences over a period of twenty-seven years.

TREATMENT

Various methods of therapy have been employed to relieve this obstinate affection.

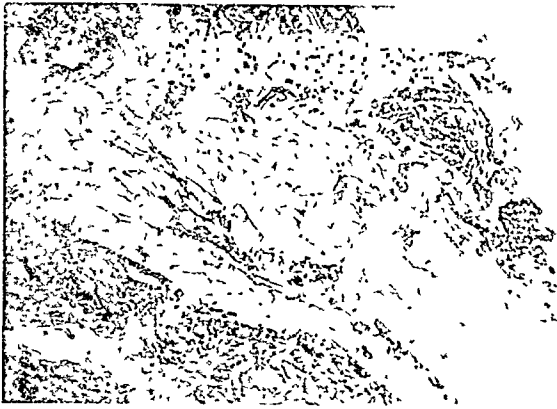


FIG. 1. Invasion of fat tissue by cellular and vascular young scar tissue.



FIG. 2. Case 1. Thick-walled artery and vein with marked perivascular round cell infiltration.

It is generally recognized that the symptoms will eventually disappear with or without treatment. Immobilization of the elbow and local applications have been used most frequently. Franke and Goedel have both resorted to operative removal of the epicondyle, but this procedure has been condemned by most writers. Fischer recommends excision of the fascia and subcutaneous tissues containing nerves. Osgood has obtained complete cures by removal of the radiohumeral bursa.

CASE REPORTS

CASE 1. L. P., male, white, aged forty-four, was first seen March 14, 1927. He stated that about four weeks previously, while working as a mechanic, he had an injury to his right elbow. While tightening a bolt with a wrench (elbow at right angle and forearm supinated) he felt a sudden sharp pain in the region of the joint. This disappeared after a few minutes, but returned when work was resumed. Following this a mild swelling was present for a few days but there was no discoloration. Pain was present whenever movement of the fingers was attempted. The pain radiated down the forearm dorsally about half way to the wrist.

Examination did not reveal any swelling or discoloration. All movements about the elbow joint were free. Extension of the wrist or fingers caused pain in the region of the external epicondyle. Supination against resistance and

full pronation were likewise painful. Very definite pressure tenderness was present over an area, several centimeters in diameter, below

the epicondyle and anterior to the head of the radius. Passive movements were painless.

The roentgen ray showed no pathology except a small bony spur at the tip of the olecranon process. This was not related to the rest of the clinical picture.

Treatment consisted of immobilization with splint, and diathermy for three weeks. During this time several carious teeth were extracted. No other foci of infection were found.

The condition remained unchanged and operation was advised.

Operation, May 14, 1927. Under local anesthesia, incision was made anterolaterally over the radiohumeral joint. This was carried down through the fascia to the extensor tendon. The conjoined tendon of the extensors was split, exposing the capsule of the joint. A bursa about 1.5 centimeters in diameter was found, anterior and lateral to the head of the radius. The edges of the bursa were surrounded by a very vascular tissue, consisting of fibrous bands and apparently newly formed granulation tissue. After opening the wall of the bursa, the tissue was found to be soft and friable and was removed in small pieces. The incision was closed in layers.

The wound healed by primary union.

Recovery was uneventful except for mild sensory disturbances along the distribution of the radial nerve. These disappeared after several weeks. They were probably due to traction or compression of the nerve by the retractor during operation.

The patient was seen at intervals for five months after the operation. He was entirely free from pain and tenderness. He had no recurrence of his former symptoms although he had resumed work.

The tissue removed at operation was examined by Dr. C. A. Hellwig, pathologist at St. Francis Hospital. Following is his report: Macroscopic: Two small fragments of grayish-red soft tissue, 4 and 5 mm. respectively in diameter. Microscopic: (See Figs. 1 and 2). No definite structure of the wall of a bursa can be made out. The specimen consists mostly of fibrous tissue and a small area of fat tissue on one side. No endothelial layer can be seen in several sections. The connective tissue fibers are extremely thick and their fibrocytes are scant. A few blood vessels are surrounded by lymphocytes. The adjacent fat tissue is invaded by fibroblasts with very fine connective tissue fibers and some capillaries.

Diagnosis: Perivascular lymphocytic infiltration. Replacement of fat tissue by young connective tissue.

Local anesthesia has very distinct advantages in a procedure of this kind. By infiltrating each layer separately before proceeding through it, the surgeon can be guided directly to the area of inflammation by the cooperation of the patient. The pathological findings, as seen under the microscope, are definite, although they are not as extensive as would be expected from the severity of the symptoms. It is quite likely that more marked changes could be seen in the granulation tissue which was present at the margins of the bursa, but unfortunately no specimen from this area was examined.

CASE II. M. B., male, white, aged forty-three, was first seen on May 19, 1927. He stated that about eight months previously, while performing heavy work with a sledge hammer, he began to have pain in the right elbow. This came on gradually over a period of three or four days, and was especially severe at night after a hard day's work. From the elbow the pain radiated down the forearm dorsally. There was also a decided weakness of the hand, most noticeable when using the hand at writing, eating, etc. Patient has kept

on working and condition has remained practically unchanged.

Examination: Range of motion in elbow is normal actively and passively. No swelling or deformity is present. A small area of tenderness is found just below the lateral epicondyle anteriorly. When forcibly hyperextending the wrist or fingers, and on full supination, a sharp pain is produced in the same area.

The roentgenographic examination revealed no pathology.

A diagnosis was made of radiohumeral bursitis. Operation was advised, but this was refused.

On December 1, 1927, patient was seen again. He had had no treatment, but kept on working. The condition of the right elbow was somewhat improved. The pain and the tenderness were less pronounced, but weakness of the fingers was still present as before.

At this time, he had in addition, a similar condition of the left elbow. This had begun about four months previously and had been the same in its onset and its course, as in the right elbow. The objective findings were very much the same as those of the right elbow.

Roentgenographic examination of both elbows was negative.

On July 8, 1928, he stated that both elbows had been healed for about four months, but that he had several recurrences since, lasting a few weeks each time.

From the clinical picture presented here, a diagnosis of radiohumeral bursitis was made in each elbow. The duration of the symptoms illustrates the course of the disease without any form of treatment.

CONCLUSIONS

1. Two cases of radiohumeral bursitis are reported. In 1, the condition was bilateral.
2. Trauma and toxemia constitute the principal etiological factors.
3. Pathological changes in the bursa are not commensurate with the severity of the clinical symptoms.
4. Treatment depends on the etiology. Where a toxemia can be demonstrated, removal of the cause would probably be sufficient. In a case which is purely traumatic, surgical removal of the bursa is indicated.

[NOTE: For Bibliography, see author's reprints]

· C A S E R E P O R T S ·

SPONTANEOUS NON-TUBERCULOUS PNEUMOTHORAX*

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COMPLICATIONS attending or following *Staphylococcus aureus* bacteriemia are so common that one tends to consider them as part of the disease. No structure or organ of the body is exempt from the local growth of the blood-borne bacteria resulting in necrosis and abscess formation. That the lungs are frequently involved is a well-recognized fact; the involvement often occurs early in the disease, allowing ample time for abscess formation, but more often as a terminal factor simulating bronchopneumonia.

In cases of early pulmonary involvement with subsequent abscess formation in which the abscess is situated just beneath the visceral pleura there may be spontaneous resolution, rupture into an adjacent bronchiole, or perforation of the visceral pleura followed by empyema. From this pathological picture one can understand the rarity of simple pneumothorax or pyopneumothorax from a subpleural abscess, which require simultaneous rupture of the abscess into a bronchiole and through the visceral pleura in order for air to make its entrance into the general pleural cavity.

It is the purpose of this report to record an instance of spontaneous simple pneumothorax from a ruptured subpleural abscess.

CASE REPORT

Long Island College Hospital no. 29-1212. E. K., a school girl aged eleven years, was admitted to the surgical service on February 12, 1929, for swelling of the left side of the

face, fever and delirium. The girl had been well until twelve days previous to admission, at which time a small pimple appeared at the left corner of the mouth. Some type of salve was applied and the pustule broke spontaneously two days later. On the third day, the mother noticed that the upper lip was slightly swollen; this, however, was unaccompanied by pain or much discomfort. Cold applications were applied locally, but the swelling increased and on the following day the father thought the patient feverish; the temperature when taken was found to be 104°F.

The family physician advised hospitalization, but this was not thought necessary until the following morning (five days after onset) when the patient's left eye was swollen shut. She was therefore taken to a hospital where she remained for one week, during which time the temperature remained between 104° and 105°F. The swelling of the left half of the face grew progressively worse, vomiting ensued and the last two days in the hospital were spent in a muttering delirium. She was taken home to die as the attending surgeon told the family that the pus had "spread to the brain" and there was no hope.

Upon admission to The Long Island College Hospital, the following positive physical findings were observed: A fairly well-nourished and slightly over-developed girl, lying in bed, restless, picking at the bed covers and muttering incoherent jargon. The skin was dry and hot, and when pinched showed evidence of marked dehydration; several petechiae were present over the extremities; the head was quite markedly asymmetrical, due to a swelling of the left half of the face which included the left half of the neck as far down as the supra-clavicular fossa. The surface of the left side of the face and neck presented a deep blue

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appearance, spotted irregularly with moderate sized pustules. The lips were pouting, the nose displaced to the right, and the left eyelids swollen to such an extent that the eye slit could not be opened manually. Emerging from the lower aspect of the swollen neck, a cord-like structure could be palpated, representing a thrombosed left external jugular vein. The right eyelids were not swollen nor were their veins dilated. No rigidity of the neck could be demonstrated. The thorax was symmetrical; the respirations were accelerated (40 per minute). The lungs were resonant throughout and nothing abnormal could be demonstrated by percussion or auscultation. The heart rate was 144 per minute, pulse regular and of good volume, apex beat in the fifth interspace. At the apex a to-and-fro friction rub could be heard. Valvular sounds were clear and no murmurs were heard. The abdomen was slightly distended; the spleen could not be felt. Findings in the extremities were unimportant except for an area over the dorsum of the right hand which was slightly raised, reddish and upon pressure caused some pain.

The Kernig sign was negative.

Blood examination:

R.B.C.	3,980,000
W.B.C.	39,700 Hgb. 77 per cent (Sahli)
P.M.N.	87 per cent
S.L.	8 per cent
L.L.	4 per cent
Trans.	1 per cent

Urinalysis:

Sp. gr. 1025

Heavy trace of albumin.

Light trace of acetone.

Shortly after admission, 5000 units of streptococcus erysipelas antitoxin were given in the left thigh muscles, and 1000 c.c. of saline were administered subcutaneously. A direct smear from two of the pustules of the face showed numerous single and clumped gram-positive organisms. On February 13, 1929, the child's condition was unchanged. A blood culture was taken and a whole blood transfusion of 320 c.c. was given with no untoward effects. Normal saline, 1000 c.c., was given subcutaneously in the morning and afternoon. The following day the general appearance was somewhat improved; the temperature remained around 104°F. and the pulse around 140 per minute. The swelling over the dorsum of the

right hand was more marked and free fluid could be demonstrated in the left knee. There was less swelling of the face.

February 15, 1929. Blood culture taken on February 13 showed 40 colonies of *Staphylococcus aureus* per cubic centimeter. The condition seemed considerably improved in the morning. Pulse rate was 120 and temperature 103.4°F. The child was rational and took fluids readily. About 10 A.M. on this day, the respirations rather suddenly increased from 35 to 50 per minute; the pulse rate rose from 120 to 148 over a period of four hours, and the temperature increased to 104°F. Examination of the thorax revealed rather distant heart sounds and absence of the previously described pericardial rub. The left thorax was limited in excursion as compared to the right. The percussion note both anteriorly and posteriorly was hyperresonant and the breath sounds were distant bronchovesicular. Roentgenograms showed a partial left pneumothorax. In the early evening, 340 c.c. of whole blood were transfused without reaction; at midnight 1500 c.c. of saline were administered subcutaneously.

February 18, 1929. During the previous two days there had been a gradual improvement with downward trend of pulse and temperature. The respiratory rate continued rapid, 45 to 50 per minute. The fluctuant area over the dorsum of the right hand was incised and about 60 c.c. of thick yellow pus evacuated. There was swelling, redness and tenderness about the left ankle and also at the distal end of the right clavicle. Whole blood transfusion of 400 c.c. was given without reaction; a blood culture was taken: Wassermann (blood serum) reaction 2 plus; white blood cells 16,000.

February 19, 1929. The fluctuant area over the upper aspect of the right shoulder was incised and about 25 c.c. of pus evacuated. There was continued improvement: less swelling of face, better appetite, temperature 102° to 103°F., pulse 110 to 130 per minute; the child took fluids readily. Pus from the dorsum of the hand showed pure culture of *Staphylococcus aureus*. Roentgenograms of the thorax showed some fluid on the left side but no apparent increase in the size of the pneumothorax.

February 20, 1929. Blood culture report taken two days previous showed 2 colonies of *Staphylococcus aureus* per cubic centimeter.

About noontime, while drinking water, the

child gave two or three gasps, became cyanotic, pulseless and died.

Autopsy B-1180. Four hours post mortem.

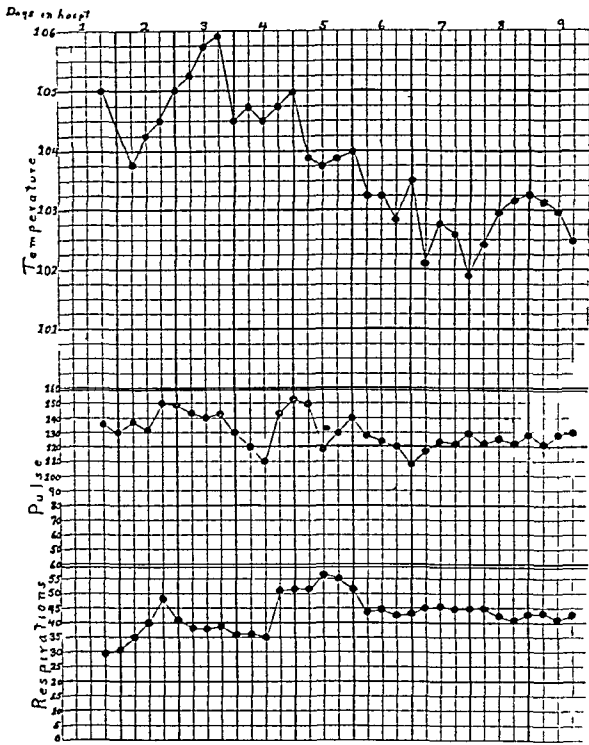


FIG. 1.

The body was that of a well-nourished and well-developed female. Over the left side of the face there was a purplish black indurated area extending from the left side of the nose to the left ear and from the left upper eyelid to the midcervical region. A thick crusty layer of dry serous exudate was present over the left upper eyelid. Surgical incisions were present over the dorsum of the right hand and the right shoulder. Both underlying cavities occupied and were limited to the subcutaneous space. The left knee was larger than the right and contained fluid.

Abdominal incision was limited to the midline. The peritoneal surfaces were free and no unusual fluid was present. (Because of the restricted incision the organs were next removed.) The entire gastrointestinal tract was negative for symptoms. The liver was normal in size, shape and color. The capsule was smooth and on multiple section no abscesses were noted. The spleen was of moderate size. The capsule was wrinkled; the pulp was soft. The malpighian corpuscles were close set and prominent. The pancreas was normal on gross examination. The kidneys were firm in con-

sistency; the capsule stripped readily leaving an engorged purplish surface. On section many abscesses were noted, measuring 0.3 to 1.2 cm.



FIG. 2.

The cut surface had the appearance of intense congestion. The adrenals appeared normal. The urinary bladder, uterus and annexae were normal. The thorax was entered through the diaphragm. Upon incision of the left diaphragm a small amount of turbid fluid escaped. The left lung was collapsed; over its upper anterior surface, 7 cm. from the apex, was a small opening in the pleura representing the center of an underlying abscess which measured 1.5 cm. in diameter. By compressing the lung, air could be expressed through the pleural opening. Situated in the apex of the lower lobe were two similar subpleural abscesses covered with a thin layer of fibrin, but not ruptured. Upon opening the right pleural cavity there was found a normally expanded lung almost entirely covered with a fibrinous exudate, the meshes of which contained a thin serous fluid. On section, three moderate-sized confluent abscesses were found in the apex of the upper lobe. The pericardial sac did not contain an excess amount of fluid. The surfaces were smooth and glistening. The pericardium over the right auricular area contained four hemorrhagic areas, the largest of these being 4 mm. in diameter. The heart muscle was firm but pale. Multiple sections revealed no areas of abscess

formation. The valves were smooth and glistening. Coronary vessels showed no evidence of thrombosis.

Microscopic Examination: Nothing except confirmation of the gross findings was elicited.

COMMENT

Literature on spontaneous pneumothorax is voluminous; mostly concerning that secondary to pulmonary tuberculosis, but very few cases have been recorded as non-traumatic, non-tuberculous in origin. Stoloff¹ has recently reviewed the literature on this subject, collecting the reports of 81 cases and adding 3 of his own. Johnson² reported 10 cases of pyopneumothorax in infants, giving a detailed account of the physical signs in this condition.

There are several points of interest in the case herein reported: Upon admission there were no physical signs elicited that would lead one to believe that there was a pulmonary lesion, with the possible exception of accelerated respiratory rate. As recorded in the progress notes and illustrated in the accompanying chart, there was a rather sudden increase in respiratory and pulse rates, unaccompanied by cyanosis, undoubtedly indicating the time of entrance of air into the left pleural cavity. The physical signs at this time

indicated decreased function of the left lung associated with diminution of the intensity of breath sounds, if not an actual change in type, whereas the respiratory rate continued throughout the remainder of the illness at a higher rate than upon admission. The pulse, temperature and physical signs indicated progressive improvement.

It is to be regretted that the autopsy had to be performed through a limited incision, for had the thoracic organ been properly exposed, the cause of sudden death might have been ascertained.

CONCLUSIONS

1. A rare complication of *Staphylococcus aureus* bacteremia, namely spontaneous pneumothorax, has been presented.
2. Typical signs of this condition were present.
3. Sudden death occurred on the twentieth day of the illness, when a favorable outcome was indicated.

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CASE REPORTS BY DR. MAURICE MELTZER*

NEW YORK

ECHINOCOCCUS CYSTS OF THE KIDNEY

I S., male, aged fifty-five years, metal worker, born in Austria, was admitted to St. Marks Hospital June 7, 1928. Patient came in acutely ill with a severe reaction following cystoscopy done elsewhere six days previously. Chief complaint before this cystoscopy was slightly blood-tinged urine of several months' duration with urinary fre-

quency ten to twelve times a day and two to three times a night. The past and personal history elicited nothing else of importance. On admission to the hospital, chief complaint was pain in the right upper quadrant, nausea and general malaise. The temperature was 102.5°F., and the pulse rate was between 105 and 120 per minute. The urine was turbid and blood tinged and urinary frequency alternated with attacks of urinary retention. Several analyses showed specific gravities

* Read before the Section of Genito-Urinary Surgery, New York Academy of Medicine, Jan. 16, 1929.

varying from 1.021 to 1.032, albumin 3 plus, red cells 2 plus and pus cells 4 plus. The white blood count was 26,000 with 92 per cent polymorphonuclears and 8 per cent lymphocytes. No eosinophiles were found. Blood chemistry showed a urea of 17.8 mg. per 100 c.c. of blood and sugar 187 mg. The systolic blood pressure was 110. The diastolic pressure was 70 and the pulse pressure was 40. Physical examination revealed a mass in the right upper quadrant extending almost to the crest of the ilium. This region was rigid and quite tender. There was pain or tenderness in either loin. Cystoscopic examination was not done because of the recent reaction.

Roentgen-ray of the urinary tract was negative except for a large shadow in the right upper quadrant corresponding to the mass described above. Because of the temperature, tenderness and rigidity of the right upper quadrant, a general surgeon was called into consultation. On June 14 a diagnosis of acute cholecystitis was made. A special examination of the biliary tract by the Graham method did not help in visualizing the gall-bladder region. An exploratory abdominal operation was advised but consent could not be obtained from the relatives.

On June 20 the patient was apparently no better and the temperature was 103°F. Cystoscopic examination was done. One hundred cubic centimeters of residual urine was found. A median bar formation at the bladder neck with diffuse cystitis was noted. Both ureters were catheterized, no obstruction was met on either side and the flow was about equal from both sides. The right specimen was very turbid. Analysis of the right kidney urine showed: Urea 0.4 per cent, pus cells (4 plus) and no casts, crystals or tubercle bacilli. From the left side the urea was 2.3 per cent, no pus, no crystals and a few granular casts. Kidney function test showed no dye from the right side and a very good dye excretion from the left side. A pyelogram was done on the right side; 25 c.c. of 12.5 per cent sodium iodide were injected. This showed an increased density in the right upper quadrant, giving the appearance of an ovoid mass extending down to the level of the 4th lumbar vertebra. The renal pelvis and its calices were not outlined. A diagnosis of a large pyonephrosis was made from the appearance of this pyelogram and from the right-sided pyuria.

An operation was advised, but the family

would not give consent. His condition gradually became worse and finally consent was given on July 10 (thirty-three days after admission). On this date the general condition was poor, the temperature was up to 105°F. and the pulse rate 130 and thready in quality. During all this interval the patient had been treated with clyses, forced fluids and the usual cardiac stimulants. On July 11 under gas-oxygen anesthesia a right nephrectomy was done. Because of the patient's poor condition the operation was quickly completed. Soon after the operation began an infusion was given. An enormously enlarged kidney, the size of a football, was removed. The next day and two days later blood transfusions of 500 c.c. were given. He did not respond to treatment and died on July 15. Postmortem examination was refused.

Grossly the kidney was about the size and shape of a football; the ureter was markedly thickened. On sectioning the kidney hundreds of various sized cysts escaped together with a very turbid fluid. The cysts varied in size from a small grape to the size of a hen's egg. Many of the cysts came out crushed and macerated. What remained of the kidney was merely a thickened pouch of atrophied parenchyma, whose contents were cysts and turbid fluid. Microscopically scoleces and hooklets characteristic of the *echinococcus* parasite were seen. Scoleces were also found in the cyst walls.

COMMENT

This patient might very well have lived had his relatives given consent for operation sooner than they did.

Echinococcus disease of the kidney is very infrequent in North American parts. In 1923, Kretchmer searched through the American literature, found 17 cases and added 1 of his own. The following questionnaire was sent out to members of the American Urological Association in order to compile the present number of reported cases. 1. How many cases of *echinococcus cysts of the kidney* have you seen? 2. Were you able to trace source of infection? 3. Was case diagnosed before operation? 4. Result of surgical procedure? 5. If postmortem examination was done, were *echinococcus* cysts found in other parts of the body?

The following table is based on the replies received to this questionnaire and includes the case reported here.

	Number of Cases
Reported	21
Seen during autopsies	3
Diagnosed by hooklets in urine	7
Diagnosed as tumors of kidney	2
Diagnosed as pyonephrosis	1
Infection traced	6
Nephrectomy was done in 16 cases:	
Cured	10
Improved	3
Died after operation	3

Of the 3 cases seen during autopsies: In 1 case the disease was present only in the kidney. In the other 2 cases, both diagnosed, disease was also found in other parts of the body.

SURGICAL POLYCYSTIC KIDNEY

E. A., male, aged forty-five, tailor, born in Hungary, was admitted to St. Marks Hospital on July 30, 1928. The chief complaints on admission were severe hematuria of several weeks' duration, vague pains in both loins, headache and general malaise. This hematuria was recurrent for the fourth time in the past four years. Two days before admission to the hospital he had been cystoscoped. A frank hematuria was found traceable to the left kidney, while the right kidney urine was clear. The analysis of the right kidney urine showed: urea 1.8 per cent, no pus, casts or crystals. The left kidney urine showed: urea 0.5 per cent, no pus, casts or crystals; red cells 4 plus (hematuria). The left-sided pyelogram showed a long and distorted pelvis and long calices, the so-called spindle-like outline suggestive of polycystic kidney. Such a diagnosis was made. The kidney function was good on the right side and there was no excretion of dye from the left side.

On admission to the hospital, cystoscopic examination was done again to check up these findings. Frank hematuria was again seen coming from the left side. A right pyelogram was done for comparison with the left side. The right pyelogram showed a decidedly smaller sized but distorted pelvis with long calices, also spindle-like in outline; the right kidney was ptosed. Both kidneys could be

palpated, the left presenting as a very large mass.

The family and personal history were irrelevant. There was no other physical sign of importance. There were no other anomalous lesions. The systolic pressure was 170, diastolic 102, and the pulse pressure 68. The blood chemistry showed a urea of 16 mg. and sugar 150 mg. per 100 c.c. of blood. The uranalysis showed a specific gravity of 1.022, a trace of albumin, no pus or sugar, and red cells 4 plus (hematuria).

On July 31, 1928, under spinal anesthesia, a large left polycystic kidney was removed. The patient had a rather stormy convalescence, but was discharged on September, 1, 1928. No hematuria has been noted since date of operation. He has gained over 15 pounds in weight. At present, the uranalysis shows a specific gravity of 1.019, no albumin, red cells, pus cells, sugar, casts or crystals. The patient complains of vague abdominal discomfort. The ptosed right kidney can be palpated. The patient is of course, under continued observation.

The pathological report of the specimen is as follows:

The kidney was 21 cm. long, 12 cm. broad, and 9 cm. thick. On sectioning the kidney, cysts of varying size were seen throughout. A hemorrhagic area (the source of his hematuria) was seen in the lower part of the pelvis. One of the cysts had apparently ruptured and produced the symptoms. The surface was covered by a thin capsule. The kidney surface was studded all over by projecting convexities varying in size from 1 cm. to a few cm. The cysts have thin walls, and are filled with pale yellow fluid, and occasionally one is filled with dark fluid. No normal kidney tissue is perceptible. On cut sections various sized cysts are seen. The pelvis is very much enlarged.

Microscopic sections are composed of small cysts; very little kidney tissue is apparent.

The following questionnaire was answered by members of the American Urological Association: 1. In how many cases of polycystic kidney have you found the condition unilateral? 2. In how many cases of polycystic kidney were the symptoms of hematuria or tumor so acute and severe on *one side* as to require surgical treatment? 3. If surgical treatment was

imperative, what was done? 4. How long have patients lived after operation?

From this questionnaire, 111 cases of surgical polycystic kidneys were compiled. The following operations were performed in this series:

	Number of Cases
Nephrectomy.	59 (53.1 per cent)
Rovsing's puncture of cysts.	31 (28.0 per cent)
Decapsulation and puncture.	5
Nephrectomy.	5
Mobilization of kidney.	2
Exploration.	4
Drainage.	2
Puncture followed by nephrectomy	1
Decapsulation.	1
Nephrolithotomy.	1

The following table shows the length of time that the patients lived after operation:

Number of Patients	Postoperative Duration of Life
4.	48 hours
24.	3 days to 6 months
20.	6 months to 2 years
44.	2 years to 8 years
3.	9 years
1.	11 years
1.	12 years
1.	13 years
2.	14 years
1	15 years
10 not followed up	

This table offers a more hopeful prognosis than usually given in textbooks and other articles on this subject.

DISCUSSION

DR. O. S. LOWSLEY: The question of polycystic kidney is a very serious one. I once removed a polycystic kidney from a doctor; I made a mistake in the diagnosis; one kidney was more affected than the other. He had such terrific bleeding that a blood clot formed, and pyelography gave us the impression that he had a tumor of the kidney, and we took it out. When I saw the surface of the kidney at the operating table I changed my diagnosis to tuberculosis, for the little cysts which showed on the surface of the kidney were not at all characteristic but looked like tubercles; there were no large cysts. Examination of the specimen when sectioned showed a typical polycystic kidney. The young doctor has not had a particularly stormy time, but has

occasional bleeding from the other kidney. That emphasizes the fact that the less one does in the way of a surgical or traumatic nature for these patients the better off they are. We had a library director as a patient who had bilateral polycystic kidneys and bleeds occasionally. We find that we can control bleeding by irrigating the pelves of the kidneys. She had a sister who died of uremia in the New York Hospital, and also had polycystic kidneys. This condition seems to run in families.

We have recently had a case of echinococcus cyst of the kidney and were fortunate in making the diagnosis beforehand, for a few of the cysts were in the bladder and were seen there with the cystoscope. It seems to be the consensus of opinion that most of the people who develop this condition are or have been associated intimately with animals. Our patient had been a shepherd in Italy, and may have had echinococcus cysts elsewhere; but so far as we know he has had no further trouble and we hope he is cured.

DR. EDWIN BEER: In reviewing 31 cases of polycystic kidneys, most of which I studied myself, I was impressed with the fact that the less one does surgically for these patients, the longer they live without symptoms of uremia developing.

In some cases stones in polycystic kidneys cause considerable distress, and must be removed. In other cases the kidneys are infected and a drainage procedure may be necessary. In those cases that bleed, it is questionable whether anything more than an intrapelvic treatment should be carried out. Nephrectomy surely is contraindicated as many of these patients are living upon a scant renal reserve. The operation advocated by Rovsing of puncturing many of the cysts, apparently leads to no results. Even the puncturing of cysts with decapsulation leads to no improvement in the kidney function.

It is remarkable how these patients run for years without any disturbance in the urinary output and blood chemistry. If the blood urea, however, begins to increase, and the phthalein and indigocarmine disappear, a serious prognosis should be entertained. In one of my earlier cases, the patient had a large left kidney tumor and the right kidney, which proved at autopsy to be large and polycystic, could not be felt. The patient was bleeding from the left kidney while the right

side delivered clear urine but no indigo-carmin, which I interpreted as reflex inhibition due to a large tumor of the opposite kidney. Under the impression that I was dealing with a hypernephroma, I exposed the left kidney under gas-ether anesthesia before the blood chemistry report had come from the laboratory. The exposure took only a few minutes. It was evident that I was dealing with a polycystic kidney, and the wound was immediately closed. Towards the end of the operation, the blood chemistry report came in showing a high retention of blood urea. Despite the fact that less than 2 ounces of ether had been used to produce anesthesia, patient became anuric and died two days after operation of uremia.

Another interesting experience in a recent polycystic kidney which was complicated by a stone that was removed by pyelotomy with some difficulty owing to the size of the kidney and the hidden position of the pelvis of the kidney, is worth recording. In this case, the stone-bearing kidney was also the seat of a rather acute infection, and the other kidney, which was also polycystic, was secreting normal clear urine. After making a primary uneventful recovery following the pyelolithotomy, the patient suddenly in the second week began to run high temperatures without any particular tenderness. The supposition, naturally, was entertained that the infection in the left kidney (the operated kidney) had flared up again, and much to my surprise on cystoscopic examination, it was found that the operated kidney at this time was delivering clear urine whereas the opposite kidney was delivering purulent urine. This puzzled us for some time until we arrived at the conclusion that possibly the bridge used under the healthy kidney at the time of operation had bruised the healthy organ and thus favored infection, a condition occasionally encountered following kidney operations.

DR. VICTOR C. PEDERSEN: I recall that four or five years ago I presented a patient with a polycystic kidney about the size of this one. I could not finish the history at that time for the woman (after we followed the policy just suggested, to leave the case alone) went on from the cold of winter to the summer and then passed out with uremia during two weeks of humid weather in August. When I presented that case no one was able to make the diagnosis. When I cut down through the anterior

route, I found a huge kidney, and no operation could have been successful. The wound was closed and the patient went home, continuing expectant medical measures until she passed away. The specimen could not be obtained.

The danger of operation is that the other kidney is also almost always polycystic. I remember a patient of this kind at the New York Hospital in the service of Dr. Robert F. Weir in the days when functional tests and roentgen-rays were in their inconclusive period. About thirty-six hours after one kidney was removed the patient died, although the risks seemed excellent. At autopsy, the other kidney was polycystic, although obviously of good function in life, but not sufficiently so to sustain operation or life alone.

DR. M. MELTZER (closing): Dr. Lowsley is quite correct in pointing out that in echinococcus disease contamination usually comes from dogs. In a hurried case presentation this point was not brought out. The infection is brought to man through contamination of vegetables or water by the infected excreta of dogs.

I was well aware of the fact that most urologists are very conservative in the treatment of polycystic kidney disease. Because such conservative opinions are entertained and because the statistics of this disease are not up to date, a questionnaire was sent out to members of the American Urological Association. The answers show that when surgical symptoms arise, many men throughout this country operate. One hundred eleven surgical cases were compiled from this questionnaire nephrectomy was done in 59 cases and Rovsing's puncture of cysts was done 31 times; in the other 21 cases such procedures as decapsulation, nephrotomy, exploration, etc. were utilized. Of the cases treated surgically 66 patients lived six months to eight years; 24 lived three days to six months. Many of these are reported as yet alive or were but recently operated. Other cases are reported to have lived nine, eleven, twelve, thirteen, fourteen, and fifteen years respectively. These figures certainly indicate that such patients are operated upon and that in many cases life is maintained for many years after such operations.

Replying to Dr. Beer the blood chemistry in the polycystic kidney showed a urea of 16.0 mg. and not 60; thus there was no evidence of retention.

The urea output from the surgical kidney was 0.5, but during a thirty-minute observation period there was no excretion of dye, thus

proving that there was no function in this kidney. The function of the opposite kidney was very good.



MULTIPLE LITHIASIS OF THE PROSTATE*

VICTOR C. PEDERSEN, M.D.

NEW YORK

DIAGNOSIS. Relapsing colon bacilluria. Multiple lithiasis of prostate. Operation.

Former General History. Good to excellent. No severe systemic illnesses. Operations for tonsils, appendicitis and piles.

Former Sexual and Urinary History. Neisser infection twenty-six years ago without complications. Prostatitis denied. Lues possible but not probable. Its history is very doubtful. The Neisser infection lasted six months under medical treatment chiefly with high pressure permanganate of potash irrigations.

Pus appeared in the urine about 1916 after the appendix operation, and at the time of leaving the hospital noted ardor and pain in urination. Urotropine prescribed five years ago on general principles and used more or less habitually ever since. Takes almost every day 2 tablets before going to bed which keeps the urine clear, pusless, pain less or absent and urination regular and normal.

Frequency of urination every four to five hours. Urgency great if not taking urotropine. Tenesmus great if not taking urotropine. No bacteriology of urine done until the case reached my hands.

General condition has remained good notwithstanding the chronic suppuration and lithiasis in the prostate and the renal involvement hereinafter shown.

External sexual organs normal. Prostate large, soft and exudes pus. No crepitation of stones.

Subcalibre Cystoscopy. 18F. cystoscope used (because standard instrument (24F.) encountered resistance in the prostatic urethra), revealed rather healthy bladder considering history and no stones.

Operation. December 27, 1927. Sacral and parasacral local anesthesia failed although 50

c.c. of 2 per cent novocaine were used. Gas and ether general anesthesia generally successful and without disturbance to the kidneys.

Median Perineal Prostatotomy (Parker Syms Operation). Lateral lobes deeply incised and nine stones removed with the finger and sharp and blunt spoons and detritus of several stones which crushed under the finger carefully washed out with high pressure irrigation.

Urethrovessical drainage tube inserted, bladder and deep field thoroughly cleansed. Bleeding decreased with 2 per cent tannic-acid solution, light packing of gauze inserted, external dressing applied. Patient returned to bed in good condition, able to speak connectedly to me before I left the hospital less than twenty minutes after closure of the wound.

After-treatment was simple. Tube out in twenty-four hours. Gauze out in forty-eight hours, sitting up on a rubber ring on the fourth day. Urinating through the normal passage somewhat on the seventh day, out in a chair on the eighth day and home on the twelfth, with the function of urination 75 per cent normal and in a condition of great comfort, wearing a small pad. Office treatment consisted in electrotherapy to the deep field of operation with great benefit.

Control of bladder absolute on January 16, nineteen days after operation.

Sailed for Europe on the twenty-eighth day after operation.

Cystoscopy and Separation of Urine. January 17, 1928. Local anesthesia of urethra and floor of bladder perfect with 10 per cent novocaine solution injected into the bladder and held by the patient and into the urethra and retained by an elastic band around the glans.

Considering long irritation of bladder the condition is remarkably good, practically

* Read before the Section of Genito-Urinary Surgery, New York Academy of Medicine, January 16, 1929.

normal as to color and bloodvessels and elasticity. The following are absent: edema, crypts, new growths, ulcers, gravel or stones.

Olive-point V. C. Pedersen multiple-eye ureteral catheters passed 25 cm. into each kidney without obstruction, pain or bleeding. Then withdrawn slightly and specimens collected.

The seven glass test showed many bacteria (chiefly staphylococci) and much pus in all glasses. Spermatozoa were in glasses 3 to 7, inclusive.

Of five specimens of urine analyzed only one was clear to the naked eye. Specific gravity ranged from 1013 to 1033. One was alkaline (catheterized). Albumen always strongly positive. Blood considerable. Pus marked and massed. Bacteria: staphylococci and bacilli (probably proteus) marked. Bacillus coli absent but present in each kidney in separated specimens.

Separated specimens were peculiarly good and similar as to all features. The bacillus coli was present in both specimens. The right urine contained 15 per cent and the left urine 21 per cent and the bladder 2 per cent in thirty minutes.

X-ray Report. Both kidneys are in good position, the right larger than the left, extending from the lower margin of the eleventh dorsal to the upper margin of the third lumbar vertebra, the left from the transverse process of the eleventh dorsal to the lower margin of the second lumbar vertebra.

There is no definite evidence of any opaque shadows in the region of kidneys or line of ureters. There are, however, several small opacities in the region of the neck of the urinary bladder, suggestive of calculi.

Whereas this patient was relieved of a major surgical condition successfully his real disease is the colon bacilluria. He arranged to have this

condition studied and relieved after his return from Europe but found himself so well at that time that he declined the plan. Thus preventive medicine was defeated.

DISCUSSION

DR. F. T. LAU: I would like to know if Dr. Pedersen found any pus present in the prostate when he operated to get the stones. I have a patient now who presents a similar picture and has much pus in his prostate; he shows two small shadows and much pus, and I hesitate very much to go in surgically for those two little shadows may be some sort of concretions rather than stones.

DR. PEDERSEN: Under prostatic examination there was much pus as shown in the seven-glass test; but at the time of operation there was a little pus but no great flow because the calculi were in separate pockets, too small for pus to show in the active bleeding. Cases of mortality following the removal of large stones from the prostate have been reported; but the feeling I had was that it would have been better to drain such pockets freely through the perineum, hoping to create freedom from infection. In the first case of this kind which I encounter, I shall certainly do that as the first of a two-stage operation, removing the stone at the second stage when infection is quiet. I should never have attempted a suprapubic operation on any of the cases reported, and not on my own case. On the twelfth day after operation he passed 75 per cent of urine through the urethra and went home; on the nineteenth day he passed all his urine normally.

I think we can approach these problems very well if we go through the perineum, and have the patient sitting up in three or four days or, when possible, sooner, for drainage by gravity.



ECTOPIC KIDNEY*

CLYDE W. COLLINGS, M.D., F.A.C.S.

NEW YORK

ECTOPIC or misplaced kidney is a rare congenital anomaly. During the past twenty years the record

toms during life and be found at autopsy. It may at times become infected, hydronephrotic, neoplastic, or calculous disease



FIG. 1. Pyelogram showing ectopic kidney and double pelvis.

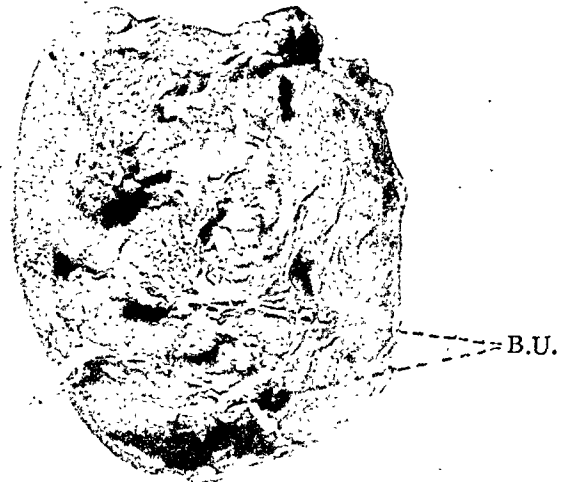


FIG. 2. Right kidney after removal. Note irregular outline of kidney mass. Actual height of mass 6 cm. B.U., bifid ureter.

room in Bellevue Hospital has not received a single case history with this diagnosis.

As Keyes¹ states, the misplaced kidney is not to be confused with the movable kidney. The latter has reached its normal position at the loin. The misplaced kidney has never reached its normal position, and is fixed in an abnormal one. It is usually on the left side, may be in the bony pelvis, or in the region of the promontory of the sacrum, the position of the kidney in this case report.

Fused or horseshoe kidney is another condition not to be confused with renal ectopia.

The ectopic kidney may give no symp-

may ensue. In each case they give the symptoms of the disease with which they are afflicted.

Mrs. R. L., aged thirty-nine, married, no children, was seen in my office November 7, 1928.

Chief Complaint: Pain in right lower quadrant.

Family History: Father living and well. Mother died during labor, brother died of "kidney trouble."

Past medical history negative.

Past Surgery: Fifteen years ago patient had a laparotomy for pain she now seeks relief from. Doctor told the patient he "scraped something inside."

Present Illness: Off and on for the past fifteen years patient has had pain in the lower abdomen, to the right of the umbilicus. This pain has been sharp at times and in the intervals a constant, dull ache. During the severe pains the patient had frequency, burning and

¹ Keyes, E. *Urology*, 1928.

* Read before the Section of Genito-Urinary Surgery, N. Y. Academy of Medicine, Feb. 20, 1929.

urgency of urination. The patient states she feels weak, and has lost 10 lb. during the past year or so.

Physical Examination: Fairly well-nourished Italian woman; appears older than thirty-nine. Not acutely ill. Examination negative except for a small, tender, cystic like mass, just to the right of the umbilicus. The mass is slightly movable. Vaginal and bimanual examination negative. B.P. 110/70.

Cystoscopic Examination: Bladder and ureter orifices normal. Left catheter up 28 cm. meeting no apparent obstruction. Right catheter up 15 cm. beyond which point it could not be introduced. Indigo-carmin, intravenously, appeared deep blue, both ureter orifices in five minutes. Pyelogram of the right kidney. Discomfort after 8 or 9 c.c. injected. This discomfort is of the same character and location the patient seeks relief from. The pyelogram showed a small, bifid kidney pelvis opposite the transverse process of the 5th lumbar vertebra. Left ureter specimen showed no pus or bacteria and 15 gm. of urea per liter, the right ureter specimen the same with 10 gm. urea per liter.

A *diagnosis* of ectopic kidney was made.

Operation: January 14, 1929. Right Nephrectomy, Pan-American Hospital, gas-oxygen anesthesia. Time of operation one hour and fifty minutes. Right rectus incision. Attempt to do the operation extraperitoneally failed due to the difficulty in locating and exposing the small kidney. Also, the great amount of perinephritis plastered the kidney down in a "bed of concrete." Transperitoneally the kidney was easily palpated, just to the

right of the vertebral column and above the promontory of the sacrum. The great number of adhesions, very short pedicle, and numerous aberrant vessels to the upper and lower poles made the operation rather difficult. Furthermore, the kidney had a double pelvis, a ureter from each pelvis passing downward for 2 inches before fusing into one canal.

Pathological Report: Bellevue Hospital No. 350/29. The kidney measures $3\frac{1}{2}$ by $2\frac{1}{2}$ by $1\frac{3}{4}$ inches. Its ureters and vessels are patent. The capsule strips easily; the parenchyma shows no abnormality.

Sections, except for the relative small size of the individual cells, show well-formed renal tissue. (A. V. St. George.)

The patient made an uneventful recovery. After being home for a week she developed a lymphatic blockage in the left leg, for which is wearing an elastic bandage. NOTE. When seen six months after operation, patient was entirely relieved.

SUMMARY

1. Ectopic kidney is a rare anomaly diagnosed by pyelography.
2. The operation is more easily performed transperitoneally.
3. Although the misplaced kidney shows a good function test it may be well to do a nephrectomy instead of freeing the kidney and a nephropexy, as advised by some. Indications for this are (a) very short pedicle; (b) possible reformation of perinephritis and adhesions about the small anomalous kidney, and return of the pain.



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EDITORIAL

SHOULD OBSTETRICS BE STANDARDIZED IN ACCREDITED HOSPITALS?*

B. H. CARROLL, M.D.

TOLEDO, OHIO

I. OBSTETRIC MORTALITY

THE mortality rate in obstetrics is ever increasing. What is wrong? Where is the cure? The rich and the very poor receive the best obstetric care, while the great majority of so-called "middle class" women continue to pay with their lives and health the high mortality rate. The American people are too intelligent to allow such a disproportion to continue unnecessarily. The public has established hospitals and given freely of money, yet the mortality rate in obstetrics is not decreasing. Where is the cure?

2. PART PLAYED BY MEDICAL PROFESSION

a. During the past two decades there has been a feverish attempt, on the part of

physicians, to advance the science of medicine. Obstetrics has shared in this "advancement," yet our mortality rate does not show it. These attempts at improvement are commendable, perhaps, if we retain and apply with the innovations the fundamentals in our field. There have been many new and ingenious modifications of instruments, changes of procedure, increased knowledge of drugs and their action. Is it a true advancement of obstetric science for the general practitioner to be informed that a dangerous drug can be packed in the nose with a degree of safety when our leading obstetricians teach the dangers of the same drug when administered in careful dosage by hypodermic? The fact remains that the dangers

* Presented as a thesis for Admission at the Forty-first Annual Meeting of the American Association of Obstetricians, Gynecologists, and Abdominal Surgeons, Toronto, Ont., Sept. 10-12, 1928.

to the mother and baby are only multiplied unless our new inventions are most intelligently applied.

In 1918 all class A medical schools required that their seniors receive at least one year's training in an accredited hospital. Did these interns, during this one year's training, receive the best in obstetric teaching or were their ideals of the class room shattered? The answer is found in our maternal death rate, which is higher now than it was ten years ago when this system of intern training was installed.

b. Procedures which are helpful. Medical schools are giving us more thoroughly prepared interns each year. The large clinics associated with the schools have demonstrated to the student, to the physicians, and to the public at large that good results can be obtained by practicing good obstetrics. The Maternal Welfare Committee has through its organization focused the attention of physicians in general to the obstetric situation, awakening a new interest in the responsibility of physicians to the pregnant woman.

3. PUBLIC

a. The action of the Maternal Welfare Committee in dividing the country into districts and placing over each district one or more physicians, interested in better obstetrics, has been a forward step in educating the public and physicians. The American Child Welfare Organization through its publications carries a note of sincerity, which will convince the people. Local lay organizations and women's clubs are beginning to discuss obstetrics. Nearly all state governments distribute free literature on maternal and child welfare. Most state laws require birth and death registrations, and nearly 90 per cent of the nation is in the registration area. Many states require certain standards, none too high, that hospitals must maintain before they are permitted to conduct a maternity service. The elimination by the state of small maternity hospitals established in old homes with inadequate facilities has

helped reduce the mortality rate. The National Government maintains its department on maternal welfare. Excellent publications and advice to the public on prenatal care are freely distributed. The passage of the Sheppard-Towner Act, right or wrong, is intended to lower the maternal death rate. With all this publicity our patients are rapidly becoming informed of the possibilities, both good and bad, which may occur during the act of reproduction. The public has been advised and still is being advised that the well-organized hospital is the safest place for confinement.

4. HOSPITALS

On the advice of physicians, the public has established and equipped many hospitals. In 1873 there were but 149 hospitals in this country. In 1923 there were 7095, an increase of 4661 per cent in the brief period of fifty years. On March 24, 1928, there were 6807 hospitals and sanitariums recognized by the American Medical Association as "a safe place for such service as they purport to render." Of this number there are 609 hospitals approved for intern training. We are rapidly reaching the saturation point for hospitals. Further development must be along the line of individual hospital improvement, and more efficient organizations of the respective services rendered.

The conduct of medical service has been left nearly in its entirety to the physicians, but let us not forget that these institutions belong to the public and must serve the needs of the public and not the convenience of the physician. So long as we direct the service in these hospitals to the best of our ability, we shall receive only co-operation from the public. The public rightly feels that the hospitals, through their administrative officers, will see to it that patients receive the best possible treatment. It is a serious obligation which we, as physicians, must help the hospital fulfill.

Another obligation the hospital must perform is the training of fifth year

medical students. The hospital is required to furnish, through designated members of the staff, "adequate instruction and experience in the various branches of medicine to these interns, especially, emphasizing obstetrics and pediatrics." If our obstetric standards in these hospitals are not in keeping with the previous instructions of these interns, we are doing the intern, the hospital, the public, and ourselves an injustice, for the young man learns a new but inferior kind of obstetrics.

5. REVIEW OF OBSTETRIC HISTORIES

a. Number of cases. After reviewing personally all of the 1927 obstetric histories from three hospitals designated for the training of interns, certain features of the service seemed to me to require further study for improvement. The total number of cases delivered were 883, all having reached the seventh month of pregnancy. Obstetricians (physicians especially interested in obstetrics) delivered approximately half or 428 of these patients. General practitioners delivered the remaining 455.

(1) Histories. Over 75 per cent of the histories were poor and incomplete. General history and physical examination were apparently hurriedly made with very little attention paid to details. Only two histories out of 455 cases delivered by the general practitioner carried notations of general pathology on the part of the mother.

(2) Obstetric histories and labor records. The obstetric history like the general history was very incomplete and the labor record far from satisfactory. Time of examinations and results of examinations were only occasionally charted. Blood pressure readings, even in toxic cases, were not always recorded. Diagnosis of labor and notes on progress of labor were very incomplete. Indications for operative procedure usually were omitted from the chart. Results of operative deliveries only casually mentioned, such as "forceps attempted then a version done."

b. Patients. Eighty-four per cent of

the patients were private cases. Primiparae outnumbered the multiparae 504 to 379.

c. Complications with results obtained. Complications were many and as might be expected rather bizarre. The complications occurring in this group of patients are not duplicated in the arrangement. For instance, an attempt at forceps followed by version will be considered as a version. Sixteen per cent of this group ended with an abnormality.

(1) High forceps were used 15 times, 2 babies died. One operation was done by an intern at the request of a general practitioner. One consultation held after delivery of patient for repair of perineum. Two mothers were infected, but both recovered.

(2) Medium forceps used 89 times; 9 babies died; 4 babies diagnosed as injured, possibly others. Seventeen mothers showed infection; temperature 101° or above for two days; all recovered. No consultations.

(3) Breech deliveries occurred 33 times, 3 babies dead, 1 diagnosed dead before delivery; 1 mother infected, recovered; 2 consultations, mother and baby normal in each case.

(4) Version with extraction was performed 26 times; 2 mothers died from shock and hemorrhage; 1 mother sustained a ruptured uterus. After the use of pituitrin, then unsuccessful forceps, version was done, baby dead. Consultation called; mother recovered, at least, sufficiently to leave the hospital alive. One mother of this group was infected, but recovered. The degree of lacerations which these mothers received was not recorded with sufficient accuracy to quote for statistical purpose. There were two third degree lacerations. Twelve babies out of 26 were dead; 1 injured, a 50 per cent mortality rate; 4 consultations were held, these mothers and babies had normal convalescence.

(5) Cesarean section was performed 18 times, 1 patient in 49; 6 of these patients had obstetric consultation and were justifiable sections. The other 12 were handled

by general practitioners and general surgeons. Four mothers were infected. Two babies died, their mothers having had one-fourth of a grain of morphine just before operation. There were no maternal deaths in this group. One vaginal cesarean section was done on an eclamptic by a surgeon and general practitioner; unable to deliver by forceps, a version was done. The baby was born dead and the mother died two hours later.

(6) Preeclampsics were 12 in number with 7 dead babies, 1 mother infected but recovered.

(7) Hemorrhage probably occurred more frequently than was noted in the history. One case which should be brought to our attention was a staff case handled by interns without supervision of staff physicians. Multipara with slow labor. Nose packed with $\frac{1}{2}$ c.c. doses of pituitrin, resulted in severe postpartum hemorrhage; both mother and baby lived.

(8) Mutilating operation on the child occurred 7 times; 4 mothers were infected making very slow recovery. One mother died after having had pituitrin, then an attempt at forceps, then an attempt at version, then forceps again. General surgeon called in consultation who did a decapitation, then a version, then pubiotomy, mother dying in twelve hours. Five consultations in this group.

(9) Labor was induced several times without consultation. Once in a case of twins, weighing four pounds and eight ounces and four pounds and ten ounces respectively; both babies died. Another case of marginal placenta previa, manual dilatation, version and extraction, mother and baby dying. This case was counted under the accidents of version. In a third case, the physician, a general practitioner, inserted a bag to induce labor. The baby's head was delivered before the bag, baby dead.

d. Normal deliveries in which complications developed.

(1) Death of mother occurred in one case due to pelvic phlebitis and abscess, six weeks postpartum.

(2) Forty-five mothers had infections from which they recovered sufficiently to leave the hospital, classified "improved."

(3) Two babies died shortly after birth, no cause given.

(4) Five babies were diagnosed as injured. There were probably more.

(5) Many cases which were apparently normal but not progressing with sufficient rapidity were given small doses, two or three minims, of pituitrin. Twenty patients received $\frac{1}{2}$ c.c. doses of pituitrin and in a few cases this was repeated several times. In this group of 20 cases, there were 7 stillborn babies. Such a shocking fetal mortality must be prevented.

e. Consultations with obstetricians were few, only 24 out of the 455 cases delivered by general practitioners. But in these cases very satisfactory results were obtained.

f. Interns. The interns handled the few staff cases in a very creditable manner, showing that they had been taught good obstetrics. Characteristic of inexperience, they attempted the spectacular at times, but in this they were aping the work of a few general practitioners. In general, the intern was not advised on the details of antepartum and postpartum care, nor the relation of complications, even minor ones, to the psychology of the patient. Scant attention was paid to the nursery, or to a study of the cause of difficult feeding. Interns in general seem to feel that the problems of the baby are a nurse's duty. The members of the obstetric staff as a whole were careless and inconsiderate of the training of the intern in such details as taking him on rounds to see the private patients, and having the intern scrub for normal or abnormal cases.

In presenting the results of this analysis of cases, the intention is not to be hypercritical of the practice of obstetrics in any one locality, but to call attention to the state of affairs which I believe exists in general in the majority of hospitals throughout the country. Two conditions must strike anyone in looking over this group of abnormalities and their management.

(1) As to the helpless young intern in such surroundings, can we expect him to build up an obstetric conscience? (2) Can we admit that these patients have been treated in the scientific manner, which they had every right to expect as they entered their hospital, when such results are recorded as are here enumerated?

In this group of patients 883 in number, 4 mothers gave their lives, 74 were infected, 57 babies died before leaving the hospital. I am thoroughly convinced that if some form of supervision had been demanded, fully 50 per cent of these lives would have been saved. After carefully studying these records from a viewpoint of prevention, I believe the following suggestions if enforced in hospitals would greatly lower the mortality rate. This then will be the first step toward the cure.

6. SUGGESTIONS FOR THE CURE

a. First, histories and examinations must be improved.

(1) The general history and complete physical examination should be placed in writing, or even better, dictated and typed on the records of all patients within twenty-four hours after entering the hospital and, unless in case of an emergency, always before delivery. The intern should have the experience of making the general examination on the pregnant woman and the working out of the details of the examination, which are at present omitted from most of the histories, but are demanded both by the American Medical Association and the American College of Surgeons requirements.

(2) The labor chart should be filled out with even greater care. Every examination whether external or internal should be carefully charted by the physician making the same, also the time of examination, and be initialed by him.

b. A predelivery diagnosis should be made and placed in writing on the chart by the examiner, together with the examiner's prognosis of the case. A written commitment will be conducive to deeper

thought on the case and a correct diagnosis made earlier in labor.

c. Division of obstetric cases into normal and abnormal is comparatively easy, when we consider that surgery is divided into major and minor divisions and no one as yet has defined satisfactorily "minor" surgery.

(1) There should be no interference in the normal private obstetric case. Staff or charity cases which are normal should be handled entirely by the intern, under the supervision of the attending staff obstetrician, usually a junior member. For self-evident reasons the staff obstetrician should be present at the time of delivery.

(2) If a predelivery diagnosis is requested in writing on the patient's chart, abnormal cases so frequently overlooked until the opportune time is passed for operation would be, in most cases, discovered at the first examination. Abnormal cases should include: (a) high forceps, (b) mid forceps, (c) breech in primiparae, (d) version, (e) cesarean section, (f) eclampsia and severe toxemia, (g) hemorrhage: before, during, or after labor, (h) mutilating operations on the child, (i) inductions of labor, (j) any general but dangerous pathology on the part of the mother which might suddenly take her life, (k) repeated doses of pituitrin, (l) postpartum infection with temperature reaching 101° or above twice in forty-eight hours.

d. The superintendent of the hospital is to be notified as soon as a diagnosis of abnormal labor has been made.

e. That consultation shall be the rule of these hospitals in all cases of abnormality.

f. Consultants should be physicians of recognized obstetric ability and sound judgment, selected to the satisfaction of the staff, the superintendent, and the Board of Trustees. Rare is the patient, knowing that she has an abnormal labor, who will object to a consultation, either for personal or financial reasons. If the

rule applied in all abnormal cases, physicians can raise very little objection to interference with their practice. If the matter of fees should arise as a barrier to this plan there are a sufficient number of good obstetricians in each community who will offer their services as consultant free of charge for six months or one year while the plan is being tried out.

g. Interns would receive training in the physical examinations of pregnant patients. A written commitment to diagnosis would sharpen their powers of observation and their ability in diagnosis. Knowing that his work would be supervised, the intern would be stimulated to better obstetric skill rather than a development in the art of bluffing. As assistant in all cases he would observe and be taught better obstetrics. If required to make rounds for private and staff cases, he would absorb many of the little details of postpartum care which have to be learned when he enters practice, often associated with unpleasant memories for both mother and doctor.

7. CONCLUSION

After reviewing the obstetric work as it is conducted in three accredited hospitals this plan to better the obstetric situation in general is presented in a spirit of constructive criticism. The hope to safeguard the mother and baby at the time of delivery, and to place around the intern on obstetric service a more wholesome atmosphere, should be our aim. It is not the normal case handled properly nor the abnormal case handled intelligently which keeps our mortality rate so high. It is meddling obstetrics on the normal case and ignorant handling of the abnormal case which causes this needless waste of life.

In trying out these suggestions it is

safe to assume that: (1) there will be no increase in our mortality rate, (2) the teaching of interns will be improved, and (3) as patients realize the protection and benefits they are to receive by this plan there will be more patients going to the hospitals for confinement.

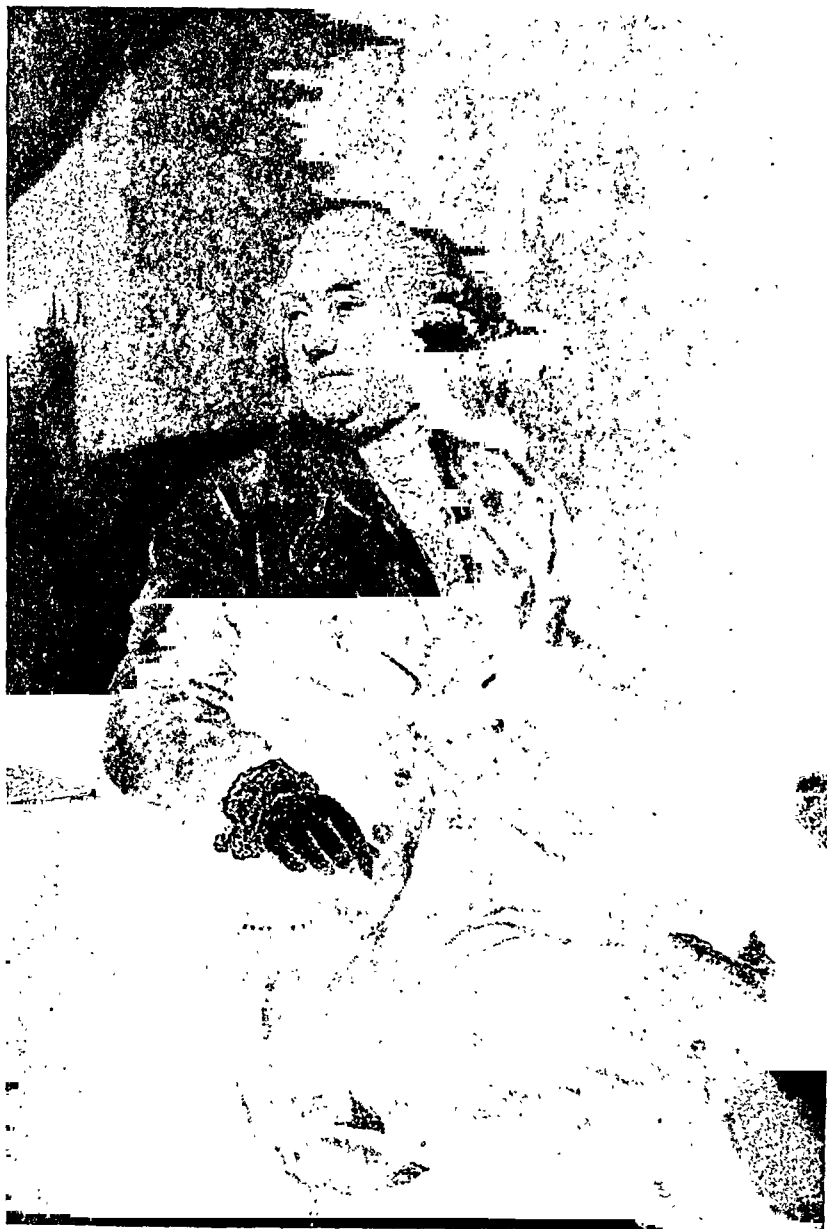
You may ask, is not this plan an unfair encroachment in the field of the general practitioner? My answer, the only reasonable answer is: "The principle, namely, to save the lives of mothers and babies, is far more important than the injured pride of any physician or group of physicians." Is not the aim and ideal of medicine to save human lives? I have enough faith in the general practitioner to feel certain that if the plan is given a fair presentation and trial that he will cooperate most enthusiastically.

If the practice of obstetrics in accredited hospitals could be standardized by this, or a similar plan, and be given the endorsement of a strong national organization backed by the American Medical Association and the American Hospital Association, a sudden check would be placed on the practice of meddling and ignorant obstetrics in our hospitals. Rightfully and logically the organization to further such a movement is the American Association of Obstetricians, Gynecologists, and Abdominal Surgeons.

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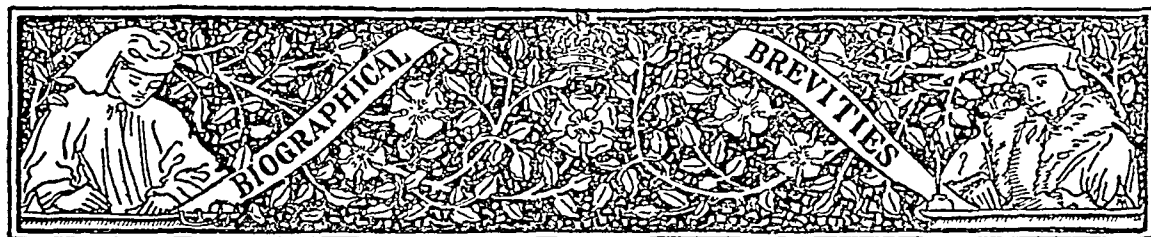
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PERCIVAL POTT, F.R.S.

[1714-1788]



“POTT’S FRACTURE”

IT is an old saying that “it’s an ill wind that blows no one good,” and this is especially true of an accident that happened to Percival Pott. He was born in 1714. He was surgeon to St. Bartholomew’s Hospital, London, from 1744 to 1787. One day he fell in the street and sustained a fracture of the fibula. While confined to his room he took to writing as a defence against ennui. Once launched upon the sea of authorship he produced rapidly and voluminously. Many of his works have been called masterpieces by discriminating authorities. In 1756 he wrote a treatise on Hernia. This was followed by others: Head Injuries in 1760, Hydrocele in 1762, Fistula in Ano in 1765, Fractures and Dislocations in 1768, an account of chimney-sweep’s cancer in 1775, and he reached his pinnacle in 1779 with his renowned brochure on caries of the spine with its resulting palsy.

Pott was a many-sided man. He had a

taste for literature. He was interested in the social life of his time. Charming in manner, kindly, considerate of others, he was the essence of the English gentleman of the 18th century. He was interested in economic questions and various charities. To the latter he gave generously.

After he passed middle life Pott had one of the largest surgical practices in London. He was a master of anatomy. He worked fast without sacrificing accurate thoroughness to speed. His personality attracted high and low alike, and his earnestness inspired confidence, which was an all-essential before the days of anesthesia, when the patient in need of surgery was in every sense a “victim.”

After more than a century and a half he has been for the most part forgotten, except for the fracture which bears his name.

Percival Pott died in 1788 at the age of seventy-four years.

T. S. W.





[From Fernellius' *Universa Medicina*, Geneva, 1679.]

BOOKSHELF BROWSING

STALKERS OF PESTILENCE

WADE W. OLIVER, M.D.

BROOKLYN, N. Y.

PART I

PREHISTORIC MAN TO GALEN

THE ANTIQUITY OF DISEASE

THE proved antiquity of disease makes it probable that illness has thrown its black shadow across human affairs ever since man emerged as man. And because man, even when most primitive, tends to pour the chances and mischances that befall him into the mould of symbol, it is quite probable that, before the dawn of recorded history, certain rudimentary concepts of the nature of disease had found their way into his brain.

In this connection, a few references¹ to the scientifically proved evidence of the antiquity of disease may not be amiss. The origin and development of disease can be traced to some extent from the pathological lesions found on the fossil bones of ancient races of man and extinct animals, as well as by pathological researches on ancient Egyptian mummies. From the researches of Sir Marc Armand Ruffer, G. Elliot Smith, Bernard Renault, Roy L. Moodie and others, it has been demonstrated that the history of disease properly begins with the early Paleozoic or Proterozoic, 100,000,000 or more years ago. The fossil remains of primitive verte-

brates of the Paleozoic Era have yielded abundant evidences of such diseases as dental caries, pyorrhea alveolaris, osteomyelitis and fracture, diseases which apparently have changed but little, if at all, in the past 100 million years. As examples, may be mentioned the fact that caries has been found in the bones, scales and teeth of fishes from the Permian period in France; the mandible of a three-toed horse shows caries which resulted in absorption of the alveolar margins and exposure of the roots of the teeth, i.e. the sequence of events which may occur in pyorrhea alveolaris as we know it today; and caries of Pleistocene elephants has been noted. One of the most interesting facts of recent development is that arthritis deformans, a disease found in so many Egyptian mummies, is identical with the "cave gout" (*Hoblengiebt*) which Virchow found in bones of prehistoric men and bears, and which also is common in the skeletons of the early German forests.

In regard to our knowledge of the existence of bacteria in prehistoric times, two methods of gaining knowledge of

such ancient bacteria have been employed: (1) actual observation of bacilli, cocci or spores in thin sections of rock and (2) by inferring their presence from results which present-day knowledge has confirmed as due to bacterial action. In 1914, Walcott actually discovered bacteria in the oldest fossil-bearing rocks of North America, found in central Montana and dating back to the Cambrian and Pre-Cambrian periods; in other words in the very dawn of the geological history of animals. The bacteria discovered by Walcott were micrococci, occurring as single cocci and in short chains; their average diameter was from $0.95\ \mu$ to 1.3μ . They were found in rocks in association with the earliest fossilized plants and animals, forms which flourished in the dawn of the world's history. Doubts that such delicate micro-organisms as bacteria are capable of preservation in a fossilized condition have been rather definitely disproved by other investigators, who have shown that fossil brains, fossil flowers, fossil blood cells, muscle and kidney structures may be so well preserved that minute details of the tissues can be accurately seen.

Renault, as a result of twenty-four years of labor, found bacteria in the petrified feces or coprolites of fishes and reptiles, as well as in fragments of bone isolated from rock which dated back to geological ages succeeding the Devonian. He also observed bacteria in the stomach contents and in the teeth and jaws of fossil prehistoric vertebrates. It is well known that certain fishes have within their rectum a spiral valve which gives to the extruded feces an irregular ellipsoidal form, with characteristic markings. These coprolites Renault found at all levels of the Autun Lake, in France, especially in certain areas, as if the fishes had lived in swarms in this prehistoric Permian lake. Frequently in sectioning a coprolite one finds remains of bones, scales and morsels of food, the residue of digestion, which have contributed to the preservation of the coprolite. In fragments of bones

and scales found in his coprolites, Renault discovered on sectioning the bones, that the lacunae and canaliculae often contained micrococci; whereas bacilli seemed to be more numerous in sections of scales than did micrococci. Renault named the micrococci *M. lepidophagus*, and described them as spherical cells ranging in diameter from 0.4μ to 3.2μ , with a tendency for the cocci to appear in the diplococcus grouping and in short chains of four members. The bacilli Renault designated *B. lepidophagus*, and described them as cylindrical rods having a length of from 4.2μ to 5.2μ and a diameter of from 0.7μ to 1.0μ . According to Renault's description of the cocci and bacilli found in these ancient bony plates, one may surmise that the destruction of bone was performed in ancient times by micrococci and bacilli whose form and proportions approach in a most remarkable manner those bacteria which we describe today as the cause of caries of bone and teeth.

In the case of the earliest known example of osteomyelitis, in the fractured vertebral spine of a Permian reptile from Texas, bacteria of the micrococcus type were found by Moodie to be abundantly preserved in the distorted osseous lacunae. They were similar in all respects to those found by Renault in the fossil bones of fishes.

PREHISTORIC MAN

The earliest prehistoric men probably had no formulated idea of the nature of disease. The hurts which they acquired during their pursuit of food, and in battles with their human and animal enemies, they probably accepted with the same unreasoning calm as they did the flicker of sunlight in front of their cave. The "why of things" had not yet started its multitudinous echoes in his brain. But, in common with the lower animals with whom he contended for his existence, the instinct of preservation was strong within him, supplemented, as it always is, by a certain rude and primitive instinct for caring for his wounds, as well as by

an instinctive hygiene. He shared with the wild beasts of the forests the instinctive hygienic lore which caused him to stretch and warm his body in the sunlight; when hurt he, too, hid in holes and caves; like the animal he, too, limped when his foot was maimed. Like the animals he perhaps sought out certain herbs and grasses when he was sick. In this sense, the statement of Le Clerc² "the first man was the first physican" is true, because instinct teaches all beings possessing sensibility the rudiments of caring for their wounds. However, it would seem that these primitive, common-sense procedures more accurately represent the original rudiments of the art of nursing, which probably preceded the medical art, and was its initiative.

As the brain of primitive man developed, all sights and sounds in nature, the rustling of leaves, the flicker of sunlight, the flash of lightning, the crash of thunder, became visible signs and manifestations of spirits, malevolent gods, or demons. In other words, he inextricably wrapped up the natural in supernatural trappings. Therefore, he worshipped the sun, the winds, the lightnings, the thunders—the era of simple nature worship. A further development in primitive man occurred when he first set up carved sticks and stones to represent these natural agencies, and thereby entered upon "fetish worship," and the practice of sacrifice to, and propitiation of, these symbols.

Primitive man, as the lowest grades of human life today, held originally to the concept that disease is an evil, extra-human spirit or the work of an evil spirit. Perhaps the next elaboration of the idea came when he began to look upon disease as the malevolent visitation of a human enemy possessing supernatural powers, whose spells and sorcery had to be met with similar weapons. And then, perhaps, as intimations of a spirit world outside the world of his daily affairs came to his awakening brain, disease began to be looked upon as the work of hostile or offended spirits of the dead.

From these primitive concepts of disease, the primitive "medicine man" and the "witch doctor" were perfectly natural and logical developments. They both assumed a solemn, and somewhat mysterious, supervisory relation to disease and its cure, not unlike that of the priest to religion. Quite generally, irrespective of geographical boundaries, the primitive "doctor" employed therapeutic procedures whose ultimate purpose was to "out-demon" the disease demons. Hence, he dressed in terrifying costumes and these, combined with his shaking of rattles and his ravings, both physical and vocal, were calculated to frighten the disease out of the patient in whom it had taken up its residence. As a prophylactic procedure, charms and amulets were provided his patients, to ward off the demon's return in the future.

ANCIENT EGYPT

Although it is to ancient Egypt that we must look for the first beginnings of the art of medicine, as well as of philosophy, it is probable that the ancient Egyptian civilization, as well as that of Mesopotamia, was nearer to, and developed more closely from, the arts of prehistoric men of the Later Stone Age than has hitherto been believed. However, the earliest known physician of which history makes mention was an ancient and celebrated Egyptian of the third dynasty (4500 B.C.), known as I-em-hetep, "He Who Cometh in Peace." He was a physician of such repute that he was later worshipped at Memphis, and a temple was erected in his honor upon the island of Philae.

In ancient Egypt, at first the priest and the physician were identified, and medicine never became fully dissociated from religion. Disease and death were believed to be not natural and inevitable, but to be caused by some malign influence which could use any agency, natural or invisible:

Often it belongs to the invisible world, and only reveals itself by the malignity of its attacks; it is a god, a spirit, the soul of a dead

man, that has cunningly entered a living person, or that throws itself upon him with irresistible violence. Once in possession of the body, the evil influence breaks the bones, sucks out the marrow, drinks the blood, gnaws the intestines and the heart, and devours the flesh. The invalid perishes according to the progress of this destructive work; and death speedily ensues, unless the evil genius can be driven out of it before it has committed irreparable damage. Whoever treats a sick person has, therefore, two equally important duties to perform. He must first discover the nature of the spirit in possession, and if necessary, its name, and then attack it, drive it out, or even destroy it. He can only succeed by powerful magic, so he must be an expert in reciting incantations, and skillful in making amulets.³

Incantations, with their implied or apparent magic element, were looked upon by the ancient Egyptians as of very great importance. In ancient days men, too, sickened and died, with much the same diseases as they do today, and the voice of the white-robed Egyptian priest comes with a pathetically reminiscent ring across the centuries as he lifted his brown arms to the sky and spoke the following magical formula: "O demon who art within the abdomen of So-and-So, son of So-and-So, O Thou whose father is surnamed He who causes heads to fall, whose name is death, whose name is the male of death, whose name is accursed to Eternity."⁴

The amulet is the oldest form of prophylaxis against disease.⁵ Originally, organs such as the brain, testicles and marrow of animals were devoured, for the purpose of reinforcing an individual's functions and his natural resisting powers. From this original concept was derived the simpler custom of wearing on the body portions of animals, or insects such as spiders, which were believed to be immune to poisons, or even rare and odoriferous objects.

Although the ancient Egyptians held to the "spirit" or "god" origin of disease, and although magic, spells, incantations and prayers held a major part in their therapeutics, yet records going back

thousands of years establish the fact that the early Egyptians employed emetics, purgatives, enemata, diuretics, diaphoretics and even bleeding as adjuvants to their psychotherapy. Later, specialism developed to such an extent that Herodotus remarked that "the country is full of physicians; one treats only the diseases of the eye; another those of the head, the teeth, the abdomen, or the internal organs."

In another connection, Diodorus remarks that so evenly ordered was the whole manner of life of the ancient Egyptians that it was as if arranged by a learned physician rather than a law-maker. Cleanliness of the person, of houses, and of cities was strictly regulated by law, and the priests, by their frequent ablutions and their spotless clothing, did much, by force of example, to stimulate interest in personal hygiene among the people.

Moreover, according to both Diodorus and Herodotus the ancient Egyptian's belief that most diseases came from overeating led to the custom of employing, for three consecutive days in each month, emetics and enemata for prophylactic reasons. It is of interest to note that the word "chemistry" is said to be derived from "Chemi" (the "Black Land"), the ancient name of Egypt, whence the science was called The "Black Art."⁶

Egyptian gods were thought to have maladies as did men. Rē had sudden diseases of the eye (thought to be symbolic of eclipses); and Horus not alone had headaches and internal pains, but he had dysentery and anal weakness as well.⁷

Worm diseases were recognized by the ancient Egyptians, a rather remarkable feat in view of the fact that even today painstaking, and often microscopic, examination is frequently necessary to establish a diagnosis. The Egyptian anemia, variously considered to be hookworm disease or bilharziosis, was thought by the ancient Egyptians to be sent by A A A, the God of Death and, according to the Papyrus Ebers, its immediate cause was ascribed to the worm Neltu.

According to Klebs,⁸ Herodotus gives an account of regulated inspection of the meat destined for sacrifices. He states that the priest, or inspector, was on the lookout for such relatively small parasitic invaders as the *Cysticercus*, and that in instances where only parts of the animal were sacrificed, the remainder was available for consumption, after a sanitary inspection had been made.

BABYLONIA AND ASSYRIA

The ancient Babylonians and Assyrians also attributed a supernatural origin to disease, believing it was caused by the presence in the body of spirits, ghosts or demons. Again it might be due to the "evil eye," or to the attack of a human enemy operating through a wizard or witch or other practitioner of black magic, or even through the agency of an offended deity. Cure of disease, therefore, depended upon the dislodgement and expulsion of the evil being by some higher, divine power; hence the treatment of disease was a matter of religion and was under the direction of the priests, who for the most part acted as physicians.⁹ Their not infrequent practice of administering to the patient foul and ill-smelling substances such as dung and urine, was apparently in order to disgust the invading demon of disease, and make his residence within the body so disagreeable that he would depart.¹⁰

The Babylonians had a large number of healing gods, to whom they sacrificed, and both the ancient Babylonians and Assyrians gave names to the individual demons of disease. Ashakku was the demon of wasting diseases; Namtar the demon of plague; Alu a demon that clouds one's vision. Jastrow¹¹ says:

The Babylonians and Assyrians thus recognized an entire faculty of demons. The age of specialization had set in which assigned a special function to each demon, although the professional ethics of demonology did not bar the demons from encroaching on the domain of a colleague.

Klebs,⁸ commenting on the fact that primitive peoples unschooled in modern scientific methods of experiment often give evidences of minute and accurate observation, mentions that on various ancient Assyrian monuments divinities are depicted in the act of manually fertilizing the flower of the date palm. In another connection, he says:

Theurgic and numinal concepts form the basis of the nosological nomenclature of the ancient Babylonians. The study of cuneiform inscriptions has opened our eyes to a new world. The texts which deal with incantations, divinations, conjurations (exorcisms) and the like contain a great deal of information on medical subjects. The priest, who filled the place of the physician, as so often in history, used these texts as a sort of practical guide-book in his daily routine. They were to lead him to diagnosis, prognosis, prophylaxis and treatment of human ills. The compilation of the most important ones can be traced to the non-semitic priests and magicians who practiced their craft before the advent of the semitic Babylonians (dated variously between 5000 and 4000 B.C. or, roughly, before 3000 B.C.).

Montaigne, in referring to the ancient Babylonians, says: "the whole people was the physican." This remark is probably explained by the following quotation from Herodotus (1:80):

They bring out their sick to the market place, for they have no physicians, then those who pass by the sick person confer with him about the disease, to discover whether they have themselves been afflicted with the same disease as the sick person, or have seen others so afflicted, thus the passers-by confer with him, and advise him to have recourse to the same treatment as that by which they escaped a similar disease, or as they have known to cure others. And they are not allowed to pass by a sick person in silence, without inquiring into the nature of his distemper.

According to the Code Hammarubi (2250 B.C.) a physician's fees in Ancient Babylonia were strictly regulated by law. Ten shekels was the statutory fee for treating a wound or opening an abscess of the eye. If the doctor caused the

patient to lose his life, or his organ of vision, the law prescribed that the physician's hands be cut off.

PAGAN SEMITES

Very little is known regarding the views of disease held by the Phoenicians and other pagan west-Semites, but theurgy, or magic, was apparently the basis of their methods of healing.

ANCIENT INDIA

In the earliest Sanskrit documents, the Rig Veda (1500 B.C.) and the Atharva Veda, we find that the Aryans of ancient India regarded disease as the manifestation of the power of some supernatural being: not infrequently arising from the gods in punishment for sin, or sometimes due to a mere caprice of a malevolent or capricious deity. Hence treatment was largely a matter of spells and incantations, hymns and sacrifices, and because often the gods were vague and uncertain, the people attributed great power to magic and sorcery in compelling the gods to obey the will of man. In ancient Hindu writings are found frequent references to the demons of disease

They are generally vague in outline and indefinite in number, and are known as *raksas* (injurer), *atrin* (eater) . . . and the like. Takman (fever), a demon, "king of diseases," was flatteringly implored to leave the body and was threatened with annihilation if he should not choose to do so. Sitala (the "Cool Lady," with an euphemistic allusion to the burning fever) was smallpox; mania a possession by *bbutas*, or ghosts; and epilepsy was the result of possession by dog-demons.¹²

In the Brahmanical period (800 B.C.—1000 A.D.) medicine was in the hands of the Brahman priests and scholars.

Hindu physiology presupposed that the vital processes are actuated by means of the air (below the navel); the bile (between the navel and the heart), and the phlegm (above the heart); from which are derived the seven proximal principles, chyle, blood, flesh, fat, bone, marrow and semen. Health consists in a

normal quantitative relationship of these primary constituents, disease in a derangement of their proper proportions.¹³

Susruta, the famous Brahman physician of the fifth century A.D., gave a very recognizable description of malaria, and attributed the disease to mosquitoes. After the Mohammedan conquest, Hindu medicine virtually ceased to exist. The great contribution of the ancient Hindus was the remarkable skill they developed in operative surgery.

ANCIENT IRAN

In the Iranian religion, reformed by Zoroaster, disease was looked upon as a diabolical entity, and was often named after the particular demon causing it. Sin and disease were on much the same plane; sin being a spiritual, and disease a bodily malady, a breach of the moral or physical order resulting from pollution, visible or invisible. This pollution must be removed by some rite of purification; hence invocations, hymns and magic must accompany the administration of natural remedies. Like the Babylonians, the Iranians worshipped, and sacrificed to a considerable number of Healing gods.

ANCIENT HEBREW MEDICINE

The Bible and the Talmud are the principal sources of our knowledge of early Hebrew medicine. In the old Testament, disease is listed as an expression of the wrath of God, and cure is to be effected by moral reform, prayers and sacrifice. In Exodus (15:26) God says: "I will put none of these diseases upon thee, which I have brought upon the Egyptians; for I am the Lord that healeth thee." Physicians were a distinct class, however, apart from the priests, and professional midwives are mentioned in Exodus. The only surgical procedure mentioned in the Bible is found in the second book of Exodus (4:25) where Zipporah, the wife of Moses, "took a sharp stone and cut off the foreskin of her son."

Klebs¹⁴ points out the fact that the

correlation between mice and bubonic plague was recognized in Biblical times, as recorded in II Samuel (2:3). After the Ark of the Covenant was captured by the Philistines and taken to Ashdod, a plague of emerods (hemorrhoids, or tumors, usually identified with bubonic plague) broke out in the town. After the plague had spread along the coast, the priests and diviners advised that the Ark be returned to the Israelites, with a trespass offering, consisting of five golden emerods and five golden mice "according to the numbers of the lords of the Philistines," to represent "images of your emerods and images of your mice that mar the land . . . to lighten his hand from off you, and from off your Gods, and from off your land." (II Samuel 6:5.)

Klebs further states: "The concepts of the insect pests among the Biblical people are largely derived from the Babylonians. Thus the fly, the mosquito and other diptera play also with them a rôle in cult and elsewhere. Ekron held an ancient and famous shrine dedicated to Baal-Zebub which name, literally translated, means the "Lord of Flies."

The Talmud, although essentially a law book dating from the second century A.D., contains some rather detailed information concerning Jewish medicine. In the Talmud is displayed considerable knowledge of anatomy, as well as of surgery, probably derived in considerable part from the fact that the ancient Hebrew religion held that the meat of diseased or injured animals was unfit food. The autopsies performed upon slaughtered animals led to some knowledge not alone of comparative anatomy, but of comparative pathology as well, and it is interesting to note that the ancient Hebrews were acquainted with caseous degeneration and tumors of the lungs, as well as with atrophy and abscess of the kidney, and cirrhosis and necrosis of the liver.

The sanitary code of the Jewish people, contained in the Old Testament, is one of the epochal contributions to sanitary

legislation, and the science of social hygiene can be said to date from it.

ANCIENT GREECE

Before Hippocrates. In the Iliad and the Odyssey of Homer, it is made evident that in the early period the Greeks looked upon disease and death as punishments inflicted on mankind by angry or vengeful gods. In the Odessey (11:411-412) is written: "It is by no means possible to avoid disease sent from Zeus; yet do thou at least pray to thy father, even unto King Poseidon." Religion in ancient Greece was an affair of rituals, not beliefs, and sacrifice was the recognized expression of piety from early times. The ancient Greeks held an intimate and friendly attitude toward their gods, and their religion and worship of the celestial deities was joyous and bright. Herakleitos said: "Men are mortal gods, and gods are immortal men." In one phase, however, their religion was dark and gloomy, namely in their worship of the black and hidden divinities of the Earth and Underworld. "These divinities ranged from Hades, the great death-god, and his consort, the dread Persephone, rulers of the Underworld realm, to demons, demigods, heroes and the vast horde of the spirits of the dead."¹⁴

As has been said, disease and death were looked upon by the ancient Greeks as caused by superhuman agencies of the Celestial World, of the Earth or of the Underworld. Disease was thought to be sent by Zeus, yet in the Odyssey (1:30) Zeus says to the immortals: "Alas! How, for sooth, do mortals reproach the gods! For they say that their evils are from us; whereas they themselves, through their own infatuation, suffer griefs beyond what is destined." In the Iliad (1:50) Apollo sends diseases and death among men; and Artemis inflicts disease, particularly mental and nervous disorders, and death among women. In the treatise of Hippocrates, "De Morbo Sacro" (The Sacred Disease) it is written: "But terrors which happen during the night, and fevers,

and delirium, and jumpings out of bed, and frightful apparitions, and fleeing away—all these they hold to be the plots of Hekate, and the invasions of the Heroes."^{1a} And in the same treatise, it is said of epileptics that

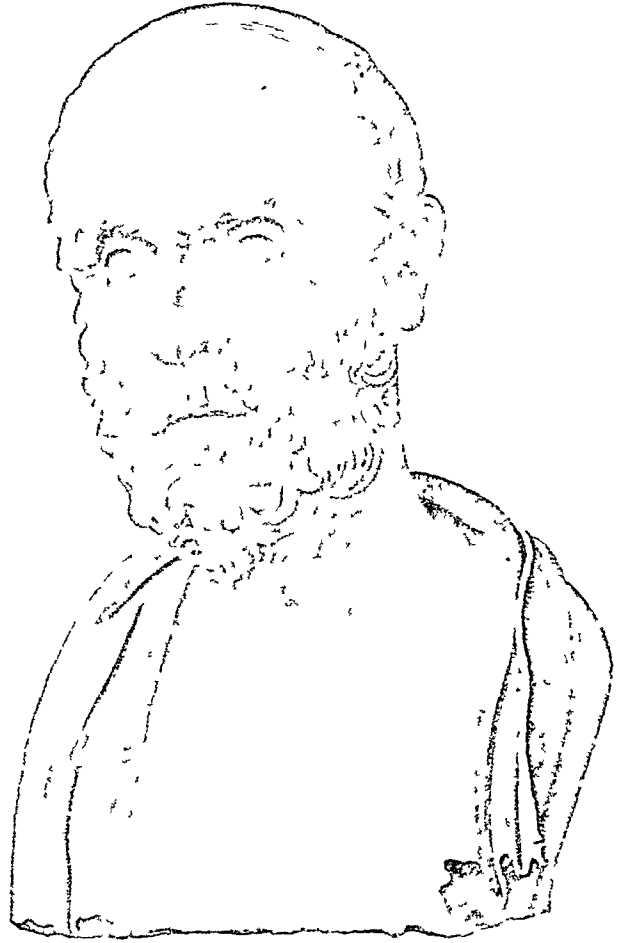
If they imitate a goat, or grind their teeth, or if their right side be convulsed, they say that the mother of the gods (Cybele) is the cause. If they speak in a sharper, shriller tone, they liken this state to a horse and say that Poseidon (Neptune) is the cause . . . but if foam be emitted by the mouth and the patient kick with his feet, Ares (Mars) gets the blame.

Sudden illness was ascribed to Pan (Euripides, *Medea*, 1170 t.f.). Contagion as the cause of the spread of disease was not recognized by the ancient Greeks. Before the time of Hippocrates, Greek medicine was regarded merely as a branch of philosophy.

The Classic Period (Age of Hippocrates). If, as a preliminary to a consideration of Hippocrates, The "Father of Medicine," we endeavor to recapitulate the development of man's ideas concerning disease from the time of early prehistoric men up until about four centuries prior to the birth of Christ, we are immediately struck by the uniformity of concept held by diverse races and peoples. There are minor variations and elaborations, it is true, but the basic thread of thought running through the minds of ancient men was that disease represented the visitation of malignant or offended spirits or gods. The element of magic was so dominant that thought and accurate observation was largely inhibited, and treatment was intrinsically a matter of ritual.

It was the peculiar distinction of Hippocrates (460-370 B.C.) that he was the first to discard the idea that disease was due to the gods or other fantastic imaginings; he dissociated medicine for the first time from religion and philosophy. This alone would have been sufficient to have insured his immortality, but he went further and by crystallizing the loose

knowledge of his predecessors and contemporaries into systematic science he became the master to whom modern medicine owes



Hippocrates
(460-370 B.C.)

the art of clinical inspection and observation. So keen were Hippocrates' powers of observation and so clear are his written descriptions of such diseases as tuberculosis, puerperal (childbirth) convulsions and epilepsy that, as has often been said, with a few changes and additions, they might be incorporated entirely in any modern text-book of medicine.

Flourishing at a time when the Athenian democracy was in full flower, a contemporary of Sophocles and Euripides, Aristophanes and Pindar, Socrates and Plato, Herodotus and Thucydides and Phidias, Hippocrates crowned his scientific labors by giving to the world his immortal "Oath" which, for over two thousand years, has

voiced the highest aspirations of the medical profession.

The incidents of Hippocrates' life are shrouded in considerable obscurity. He was believed to have been either nineteenth or seventeenth in direct descent from Aesculapius and, according to Soranus, was born in Cos. He is said to have studied medicine under Heraclides, his father, and Herodicus of Selymbria, and philosophy under Democritus of Abdera, the originator of the doctrine of atoms. He taught and practiced medicine in Athens, as well as probably in Thrace, Thessaly, Delos and Cos. He died at Larissa in Thessaly, at an age variously estimated from eighty-five to one hundred and nine years.

The broad basis of reasoning upon which Hippocrates built his system of medicine is profitably to be considered before a closer analysis of his views concerning the nature of disease is attempted. Although a member of the family of the Asclepiadae, priest-physicians steeped in traditions of magic and priestcraft, the bed-rock of his contention was that medicine must be dissociated from priestcraft and philosophy. In his "de Aere," (29) he expressly states that attention should exclusively be directed to the natural history of disease and that diseases must be scientifically treated as subject to natural laws. Charms, amulets, incantations were anathema to him, trappings of an outworn magic which has no place in medicine. Here we have enunciated the "Magna Charta" of medicine, and to Hippocrates can rightfully be given the title of the "Emancipator of Medicine" from superstition. It is no proper reflection upon his genius that some of his medical descendants, ancient as well as modern, attempted to rewed medicine and magic.

Hippocrates, intellectual giant that he was, was handicapped by certain definite limitations of knowledge. The sacred veneration which the ancient Greeks felt for a dead body precluded dissections, and hence the knowledge of anatomy, physiology and pathology possessed by

Hippocrates was incomplete and confused. He seemed to have no conception of the infectiousness of epidemic diseases, as we know it today, and he attached a quite erroneous importance to "miasmas" as a cause of disease. In his book entitled "On Winds" he wrote: "According to all appearance the cause of disease should be found in the air, when it enters the body in excess, or in insufficient quantity, or too much at a time or when tainted by morbid miasmas." In his book on "Humors" we find: "Diseases develop from odours exhaled from mire and marshes," and in his "On the Nature of Man" Hippocrates states that the cause of epidemic diseases resides "in the air from which escapes a morbid exhalation contained within it" and he lists the air as the only possible "universally acting cause" of wide spread disease. And yet Hippocrates seemed to have rather definite ideas regarding infection from water, for we find that not alone did he advise that water be boiled before it was drunk, but we find him writing in his famous treatise "On Airs, Waters and Places" the following:

The still and stagnant waters of marshes are the cause of diarrhea, dysentery and intermittent fever (malaria). Those cities which are favorably placed for the sun and the winds, and where the waters are of good quality, are less touched by these disadvantages. But those where stagnant and marshy waters are used, and whose site is bad, suffer more from them.

It is interesting to note, as pointed out by Singer¹⁶ that Thucydides (B.C. 471-391), a contemporary of Hippocrates, was the first writer to whom can be traced a definite and formal belief in the passage of specific infectious disease from one person to another. In his "History of the Peloponnesian War," (11:47 ff.) he says, in speaking of the plague of Athens, that those who came most intimately in contact with the sick were the most liable to contract the disease. Thus we find that, among the ancient Greeks, as well as the ancient Romans, although the wide-

spread nature of epidemics was largely a mystery, yet the effects of excessive rainfall, certain winds, the seasons, overcrowding and the inhalation of air breathed by others, as well as unburied corpses, were all considered dangerous to health. However, with the ancients, it was miasma rather than contagion that was most feared.

Although the central idea of Hippocrates' humoral pathology in regard to the causation of disease has largely been discarded, yet it marked a distinct advance over concepts hitherto held by its insistence that disease resulted from a derangement of certain fluids of the body, brought about by natural laws that are subject to no magical influence. The monumental advance made by Hippocrates in his concepts of disease becomes the more graphic when one realizes that the very essence of his teaching was the existence of a formative, conservative and curative power in the human body, by which it preserves itself and combats morbid causes of disease, and the effects produced by them. Its scientific virtue lay in centralizing attention upon bodily changes, a virtue not vitiated by numerous errors of detail which are incorporated in Hippocrates' theory. It postulated that, corresponding to the four elements (fire, air, water and earth) and the four qualities (hot, cold, moist and dry) there are four humors or fluids of the body, viz. blood, phlegm, yellow bile and black bile. Health was considered to depend upon a proper balance of these humors, and disease upon an imbalance. Hippocrates taught that such general causes of disease as heredity, climate, the seasons, and the epidemical constitution acted by disturbing the equilibrium of the body, specifically by changing the humors. For example, fever results from a heating of the bile or from an increased production of phlegm which causes the tissues to swell, thereby interfering with the secretions; a chill was considered to be an indication that phlegm had become mixed with the blood. The fermentation or putrefaction

of the humors, from which fever and contagion were believed by the Hippocratic School to arise, might manifest itself externally, as in various skin diseases and in septic sore throats, or it might be confined to the internal structures of the body, and not become visible.

The tremendous influence which Hippocrates exerted upon medical thought is amply attested by the fact that his doctrine of disease survived, with minor modifications, for almost twenty-two centuries. The masterful clinical grasp of Hippocrates, largely made possible by his powers of minute observation and analysis, are abundantly testified in his writings. In his book on "The Uses of Liquids" he observes that when fishermen suffer wounds, suppuration does not tend to occur, unless the wounds are meddled with; and he extols the anti-putrefaction properties of sea-water. Infectious processes following wounds, such as pyemia, erysipelas and tetanus were accurately described by Hippocrates, as well as infections following labor and miscarriage. In his "Diseases of Women" he writes:

If the womb is ulcerated, blood, pus and ichorous fluid are discharged . . . the belly becomes distended . . . and it is painful to touch as is a wound. Fever, grinding of the teeth, continued pain in the genital parts, pubis, lower abdomen, flanks and lumbar region ensue; this disease is especially prone to occur after a labour when something putrefies in the womb; it also occurs after abortion.

In the books of the "Epidemics," Hippocrates details a case of a wound of the skull with splinters of bone which required trephining. He tells how pus formed and how, on the eighth day, a chill occurred, followed by fever. He points out that the fever and chills are due to the accumulation of pus and he recommends a thorough cleaning out of the wound by a surgeon as the only means of saving the patient's life. In the same book he refers to a case of abdominal suppuration in which cauterization stopped the discharge of pus, but because of a mistake in diet

during very warm weather fever and diarrhea developed, and the patient died. It is interesting to observe that Hippocrates not only resorted to heat for destroying the focus of pus, but he attached great importance to the patient's resistance, and comments on the fact that the good effects from his operative procedure were vitiated by the patient's indiscretion in the matter of diet. Hippocrates also recognized the connection between pus and fever, because he writes: "The majority of patients who have suppurative processes are seized with chills and fever."

After the death of Hippocrates, his son-in-law Polybus carried on the torch of medicine which he had lighted, as did Dioxippus of Cos and Petronius and Diocles, the latter being called by the Athenians "the second Hippocrates." The School of Alexandria, resulting from the colonization of Greek medicine in Egypt, is particularly notable for its anatomical discoveries which so profoundly influenced ancient medicine. Of this school, the most famous leaders were Herophilus and Erasistratus (300-225 B.C.), the famous anatomists who are said to have originated dissecting. The activities of the Alexandrian School were concerned with more or less minor modifications of Hippocrates' methods of treatment.

THE GRAECO-ROMAN PERIOD

(146 B.C. - 476 A.D.)

Before Galen. Was it Carlyle or Emerson who said: "Beware when God lets loose a thinker on the world, for all things then have to rearrange themselves?" An Hippocrates has some such action in changing the latent but unorganized possession of facts to definite conscious possession of truth as does a crystal added to a supersaturated solution. Not alone that, but a ferment is set in other men's minds, a catalyzer of the intellect that activates otherwise latent and slumbering thought processes. Before Hippocrates, the supernatural trappings with which disease had

been invested had served to inhibit men's imaginings in regard to the nature of disease. After Hippocrates, the floodgates of theorizing opened. Following his lead, attention became centralized upon bodily alterations as constituting the essence of morbid processes, but theory unsupported by experimental facts led medicine during the major part of the Graeco-Roman period into cul-de-sacs of sterile dialectic.

Thus we find Asclepiades, born in 124 B.C., advancing an "atomic" or "corpuscular" theory of disease, based on the teachings of Epicurus. He taught that the human body, like other matter, is composed of small, unchangeable and individual atoms, the soul also consisting of atoms in a more subtle state. Illustrative of the ingenious, but misguided lengths to which he went is the following quotation from Cumston.¹⁷

Asclepiades states that the human body remains in its natural state as long as matter is freely absorbed between the pores, the interatomic spaces, so that health results from a correct proportion of pores in relation to the matter which they should absorb and allow to pass. Death is a result of a disproportion between the pores and matter. The most common accident is obstruction of these pores, which, according to Asclepiades, produces phrensy, lethargy, pleurisy, high fever and pain. If the pores are too oblique, the result will be the production of fainting, languor, exhaustion and so forth, while extreme emaciation and hydropsy are the result of extreme dilatation of the pores. Hunger is engendered by opening of the large pores of the stomach and abdomen, while thirst is due to opening of the smaller ones. Finally, from a rather obscure passage in Caelius Aurelianus, it would appear that Asclepiades admitted a third cause of disease, namely, a disturbance or confusion of the body juices, or the mixture of liquid matter with the spirits. He explained intermittent fever (malaria) as follows: The quotidian is produced by the retention of the largest of all the atoms, the tertian by that of the medium atoms, and the quartan by that of the smallest.

Asclepiades discarded the idea that there are any specific remedies for the various

organs of the body, and he ridiculed the passiveness of the Hippocratic School, saying: "the practice of the ancient physicians was merely a meditation on death." The so-called Empiric School of Medicine, of which he was the leader, has gone into the limbo of historical curiosities, but Asclepiades remains of interest because of the fact that he was the first to mention tracheotomy.

The main contribution of Themison (123-43 B.C.) who practiced medicine in Rome during the reign of Augustus, was his division of diseases into two classes, *the acute and the chronic*. Founder of the so-called Methodic School, he modified the "pore" theory of Asclepiades by teaching that the principal causes of disease were (1) a constriction (*strictum*) of the pores of the body, (2) a relaxation (*laxum*) of the pores, and (3) a mixed cause, resulting from a constriction of the pores in one part of the body and a relaxation of the pores in other parts. Themison followed Hippocrates in distinguishing three periods in the evolution of disease: (1) a period of increase, (2) a period of full development, and (3) a period of decline.

Thessalus (A.D. 60), who also practiced in Rome, introduced a still further modification of the "pore" theory. Whereas Themison and Asclepiades had taught that health resulted from a symmetry of proportion of the pores, and disease from a disproportion, Thessalus believed that in order to cure a disease the condition of the pores in the diseased part should be entirely changed, and from this theory is derived the word "metasyncrisis," which signifies a change in the pores. Mustard was a favorite metasyncritic drug employed by Thessalus. Not content with damning Hippocrates by characterizing his "Aphorisms" as a tissue of lies, and with stating that all physicians who had lived before him had done nothing useful, Thessalus made a pathetically ridiculous gesture to posterity by ordering that upon his tomb should be engraved the words: "The Conqueror of Physicians."

Methodism, founded by Themison, continued to be the most important of medical sects during the second century A.D., at least in Rome, and during this century its two most distinguished champions were Soranus of Ephesus, the greatest gynecologist of antiquity, and Caelius Aurelianus, a contemporary of Galen. Both Soranus and Caelius held strongly to the pore constriction and pore relaxation idea of the causation of disease, and they divided the principal types of acute diseases into three main categories: (1) phrensy (ataxic fever) and lethargy (adynamic fever) result from a constriction of the pores; (2) cardiac disease is due to relaxation of the pores, and (3) pneumonia and pleurisy are in the mixed type, i.e. these diseases result from a constriction of the pores in certain parts of the body and a relaxation of the pores in other parts. Among chronic diseases, vertigo, asthma, epilepsy, melancholia, jaundice, paralysis, catarrhs, phthisis, colics and dysentery are due to constriction of the pores (Caelius); diarrhea and hemorrhagic and menstrual flux, when excessive, are due to relaxation of the pores; whereas hydropsy belongs to the mixed type.¹⁸ Caelius was an ardent champion of the belief that no such thing as a local disease exists; he contended that when a person is ill, the entire body is diseased, an opinion that is considered today the ultramodern conception of such a disease as pneumonia, which is looked upon as a disease of the entire body, with marked localization in the lung. From this, Soranus held the extreme opinion that the search for the organ principally diseased is a bootless task, and both Soranus and Caelius denied that there were any specific drugs, i.e. drugs having a particular action on a given organ.

During the second century A.D. Aretaeus the Cappadocian extended the theory of infection advanced by Thucydides. In his volume on "The Causes and Symptoms of Acute Diseases," he distinguished definitely between contact infection and infection at a distance.

Galen and the Restoration of Hippocratic Medicine. Medical thought during the Graeco-Roman period was characterized by rebellion against the teachings of Hippocrates and a grotesque theorizing upon the nature of disease, unsupported by a sufficient body of facts. The advent of Galen marked a return to Hippocratic medicine.

Galen, whose full name was Claudius Galenus, flourished from 130 to 200 A.D. After studying medicine in Greece and Egypt, he came to Rome to practice when scarcely thirty years of age. He was contemptuous of the various sects, such as the Methodists, Empirics, etc. and he accepted the teachings and precepts of only one master, Hippocrates. Equipped with an extremely active mind whose tendency was always to explain facts by hypotheses, and possessing an encyclopedic knowledge of medicine, Galen did not so much advance man's knowledge of the nature of disease as to refurbish the Hippocratic doctrines in more popular and attractive trappings. This statement would seem to hold true in spite of the fact that Galen was not alone the supreme anatomist up to the time of Leonardo Da Vinci (1510) and Vesalius (1543), but certain of his discoveries would seem to entitle him to consideration as the first great experimental clinician. He demonstrated the difference between the motor and the sensory nerves, and even distinguished the motor and sensory roots leaving the spinal cord; he also recognized the difference between intercostal and diaphragmatic breathing.

Like Hippocrates, Galen recognized four elements (fire, water, earth and air) and their four qualities (heat, moisture, cold and dryness) as constituting the basis of all parts of the body. He taught that the body consists of three distinct parts: (1) the solid parts, such as bones, ligaments various organs, etc., which he likened to containers; (2) the liquid parts or contents, such as the blood, lymph and bile; and (3) spirits, or forces. These latter "spirits" he believed to be three in number (a)

natural spirits, which were a subtle vapor arising from the blood (which, itself, was formed in the liver); (b) vital spirits, which were formed by natural spirits going to the heart and there combining with air; and (c) animal spirits, which resulted from the transformation of vital spirits in the brain. (Parenthetically, it is to Galen that the doctrine of Vitalism, still debated in physiology and biology, is to be traced.)

Galen, moreover, admitted four principal body humors: (1) the blood, a hot and moist humor, (2) pituit, or lymph, a cold and moist humor, (3) yellow bile, a hot and dry humor, and (4) black bile, a cold and dry humor. Most diseases, he held, arose from an excess, or deficiency, or an alteration in these four fundamental humors and their specific qualities. Perfect health, on the other hand, occurs only when complete equilibrium exists between the four humors and their four qualities.

It was from these concepts that Galen built up his classification of temperaments, which he subdivided into four primary types: (1) the sanguine, (2) the phlegmatic, (3) the bilious and (4) the melancholic. Each of these temperaments he believed to be either hot, or cold, or moist or dry. Differences between temperaments, and their divergence from the normal, he looked upon as predisposing causes of disease; the exciting causes being a disordering of the humoral equilibrium. Hence, each disease demands its particular curative treatment; in one disease the humors must be thinned; in another thickened; in one cooled, in another warmed; in one purified, and in another evacuated. Galen was an ardent advocate of bloodletting, but he said it was better to err on the side of insufficient bloodletting than of excess. Galen's idea that "coction" or suppuration is an essential part of the healing of a wound gave rise to the theory of "laudable pus," an error which was not completely dispelled until the researches of Lister and Pasteur, in the latter half of the past century.

Galen followed the Hippocratic school

in attributing the cause of epidemics to atmospheric conditions; he emphasized the effect of climate, as well as the degradation of the air, this latter being brought about most commonly by swamps and stagnant waters.

Galen was the most voluminous of all the ancient writers and the greatest of the theorists and systematists. His works are a gigantic encyclopedia of the knowledge of his time, including nine books on anatomy and seventeen books on physiology (apparently, Galen was the first to dissect the muscles and describe them, probably by dissection of apes, and his writings give us the most complete picture of the anatomy and the physiology of the human body of any works handed down from antiquity); six books on pathology, sixteen essays on the pulse, the "Megatechne (Ars magna)" or therapeutics, in fourteen books; the "Microtechne (Ars parva)" or practice, and thirty books on pharmacy.

The domination which Galenic medicine held over the thoughts of men during the major part of the Christian era presents an interesting psychological problem. He constituted the Delphic oracle of medicine, and his pronouncements upon all matters relating to disease became surrounded with a false aura of infallibility. But time has dealt harshly with Galen's theories, and in the light of modern experimental medicine his hypotheses and system of medicine have been completely overthrown. On the other hand Hippocrates, with his unflinching insistence on the value of accurate, painstaking observation and his impatience with theorizing, has grown in stature as the centuries have passed.

ANCIENT CHINESE THEORIES OF DISEASE

It has been said that medicine in China reached its highest development at about 2000 B.C. and has remained at about the same level up until very recent times. Chinese medicine is of practical interest mainly because of certain therapeutic measures which were introduced by the Chinese into Western medicine, among

these being dry cupping, massage and acupuncture. The ancient Chinese are said to have practiced preventive inoculation against smallpox, as well as to have employed mercury for inunction in syphilis. According to Read,¹⁹ the art of pulse-feeling in China is very ancient, dating from before 500 B.C.:

By placing three fingers side by side on the left and right wrists respectively of the patient, the Chinese physician claims to be able to obtain six indications of possible diseases in the internal organs . . . In general, the left wrist indicates diseases of the kidneys, the heart and the sexual life. The right is felt for the lungs and so forth.

Dawson's²⁰ article, concerned with a previously untranslated work, "Su-Wen" or Simple Questions, by an unknown Chinese writer who probably lived prior to the end of the fourth century A.D., is of especial interest because it sums up the physiological and medical knowledge of all the preceding centuries. Curiously enough, the theories have been little, or not at all, modified and they form the foundations of present day Chinese physiology and psychology. Man is pictured as containing five viscera: (1) the heart, the seat of the vital spirit; (2) the liver, the seat of the aerial soul, which directs the internal distribution of the breath; (3) the lungs, the seat of the spermiatic soul; (4) the spleen, the seat of the intelligence and of the will, and (5) the kidney, the seat of the fecundating sperm, and the procreating organ.

Disease is thought to come from a functional disorder of a viscus, and the only method for determining the sick viscus is by an examination of the pulse. Each variety of pulse (i.e. slowness, rapidity, weakness, etc.) is taught to be pathognomonic of the disfunction of a given viscus. When the sick organ has been diagnosed, dietary measures are immediately to be instituted. For example, if the heart is affected, it is because the patient has overindulged in hot and bitter foods; if the kidney is diseased, it means that

cold and salty foods have been used to excess.

Dawson states:

Finally, let us speak of therapeutics. Here I call attention to an important fact. Before the Christian era the Chinese did not recognize and did not use medicaments properly speaking. So then, in ancient times, there were no medicaments properly so called, but a unique proceeding believed to heal all ills, namely the famous Chinese acupuncture. Antiquity knows only this: The invention probably dates from primitivity, for the needles for a long time were of flint. Here is the theory of acupuncture: . . . Every malady is a visceral disorder. A viscus either does or does not secrete its emanation, or does not secrete enough of it, or retains it instead of circulating it. In a word paralysis of the organ, bad work . . . Now, what does one do to the ox when he draws badly? One gives him a blow with a goad. The Chinese acupuncture is nothing else. One pricks the recalcitrant organ. The Su-Wen gives the instructions for these direct pricks, but it also indicates rather well the inconveniences and the danger of the method, if one uses the needle too violently, if one pricks ever so little to the side and so forth. The direct prick should be reserved for desperate cases, when there is nothing to lose.

In ordinary cases, indirect acupuncture should be practiced, which consists in pricking the skin area, or cutaneous district, over which a given viscus is supposed to preside:

For example, if the pulse has incriminated the heart as the offending viscus, a certain cutaneous region of the back corresponding to the heart is pricked, whereby the heart frisks with pain and then settles down to its normal functioning.

(To Be Continued)

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BOOK REVIEWS

THE NOSE, THROAT AND EAR AND THEIR DISEASES. Ed. by Chevalier Jackson, M.D., SC.D., LL.D., F.A.C.S., George Morrison Coates, M.D., F.A.C.S., assisted by Chevalier L. Jackson, M.D. Phila., W. B. Saunders Co., 1929.

This book by 74 contributors has the defects of its qualities, a fact which is faced by the Editors who state in the preface: "In a systematic work by many authors one does not expect to find the degree of consistency usual in a book written entirely by one author. Indeed it is one of the advantages of such a work that conflicting opinions by leading authorities may be presented. The very complete index will refer the reader to different articles in which certain subjects are separately considered by two or more authors from radically different points of view." This makes it a splendid reference work for the general practitioner but as to the wisdom of placing all the different viewpoints before students there may be much difference of opinion and it is likely that this book will be more popular with the general practitioners and medical men than as a textbook for the students. The need for such a book has long been felt and this book, written by the leading men in the country and edited by Chevalier Jackson, will it is hoped fulfill this need. Appended is a list of the contributors. The subjects treated are thoroughly covered. As is to be expected the articles

are of varying value. The references for reading are very good as far as they go but by no means complete. The illustrations are numerous and well chosen and the bookmaking is excellent.

Contributors: Arthur M. Alden, Edmond Aucoin, James A. Babbitt, Harry A. Barnes, John F. Barnhill, Joseph C. Beck, Herbert Stanley Birkett, Albert P. Brubaker, W. M. C. Bryan, Carl J. Bucher, Ralph Butler, Harry Philip Cahill, Thomas Edward Carmody, William B. Chamberlin, Louis H. Clerf, George Morrison Coates, Lewis A. Coffin, Baxter L. Crawford, Ewing Wilbur Day, David Bryson Delavan, John Kolbe Milne Dickie, Wells P. Eagleton, Francis P. Emerson, Ebenezer Ross Faulkner, Ralph A. Fenton, George Fetterolf, Thomas Rushmore French, Elmer H. Funk, Perry Gladstone Goldsmith, Max A. Goldstein, John M. Ingersoll, Chevalier Jackson, Chevalier Lawrence Jackson, Fernand Lemaitre, Eugene Richards Lewis, Fielding O. Lewis, Robert M. Lukens, R. C. Lynch, John Edmund Mackenty, Robert Maduro, Willis F. Manges, Emil Mayer, Irwin Moore, William F. Moore, Harris P. Mosher, Emile Moure, William V. Mullin, Charles F. Nassau, Francis R. Packard, John R. Page, John B. Rae, B. Alex. Randall, Henry O. Reik, William Scott Renner, George L. Richards, Lyman G. Richards, Robert F. Ridpath, J. Parsons Schaeffer, Edward Cecil Sewall, George Elmer Shambaugh, Burt Russell Shurly, Harmon Smith, S. MacCuen Smith, Robert Sonnenschein, Frank R. Spencer, Henry L. Swain, Clement F. Theisen, Sir St. Clair Thomson, Gabriel Tucket, A. Logan Turner, D. Harold Walker, I. Chandler Walker, Leon E. White, and George B. Wood.

PRINCIPLES AND PRACTICE OF ELECTROCARDIOGRAPHY. By Carl J. Wiggers, M.D. St. Louis, C. V. Mosby Co., 1929.

As would be expected, Dr. Wiggers has written a clear-cut, concise and yet complete book on the principles and practice of electrocardiography. It is interesting to note that this is the third splendid American production on this subject during the past year, a further indication that we are no longer dependent on Europe for our monographs.

OSTEOMYELITIS AND COMPOUND FRACTURES. By H. Winnett Orr, M.D., F.A.C.S. St. Louis, C. V. Mosby Co., 1929.

Dr. Orr's stimulating writings of the past years have created a demand for a book cover-

ing his individual work. This demand is thoroughly met in this splendid little volume which is compiled in a practical manner, stressing all the points that Dr. Orr has previously made and that heretofore have been scattered through the literature. While every one will not agree with or want to use all of Dr. Orr's methods, every surgeon in the country should read the author's presentation of his subject and we venture to prophesy that many who have not formerly used his methods will do so after reading this book.

COLLECTED PAPERS OF THE MAYO CLINIC AND THE MAYO FOUNDATION. Ed. by M. H. Mellish, Richard M. Hewitt, M.D., and Mildred A. Felker, B.S. Phila., W. B. Saunders Co., 1929.

This is the twentieth volume of the collected papers of the Mayo Clinic. It is interesting to note that in order to keep the annual literary output of this famous institution within the limits of a twelve hundred page volume it has been necessary in 233 instances to give only references. Forty-three are published in an abridged form, 72 abstracted and 81 (of course the cream of the output) are published complete. An invaluable part of this volume naturally is the index, enabling one to refer at once to the latest word from the Mayo Clinic on any given subject. The publishers have done their work well but particular credit is due to the editorial staff of the Mayo Clinic.

PROCTOLOGY. A Treatise on the Malformations, Injuries and Diseases of the Rectum, Anus and Pelvic Colon. By Frank C. Yeomans, M.D., F.A.C.S. N. Y., D. Appleton & Co., 1929.

Considered from every angle, that of the specialist, the internist and the general practitioner, Dr. Yeomans' book is by far the best we have been privileged to study. Other books on proctology have been written, many of them excellent and beyond criticism, except that one might venture to assert they are too large, contain too much irrelevant material, are not designed for the man in the field, or, on the other hand, that they are too small, monographic in character and dwell in detail on but one fragment of the large subject.

Dr. Yeomans has done a splendid bit of work. He has fashioned a book that is readable and without padding. There are many (417) illustrations and they are exceptionally good. There are 35 chapters and each one has many sub-divisions.

We sincerely and earnestly recommend this book to every student of medicine and to every practicing physician and surgeon.

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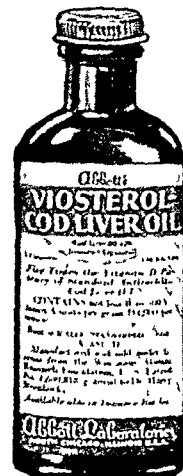
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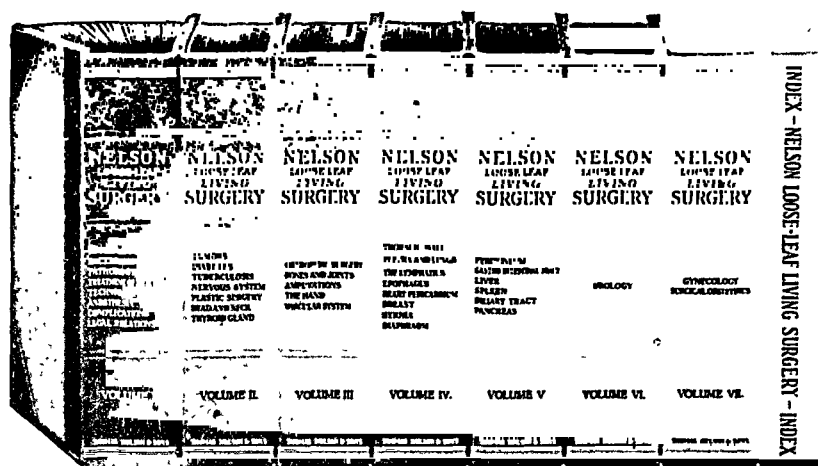
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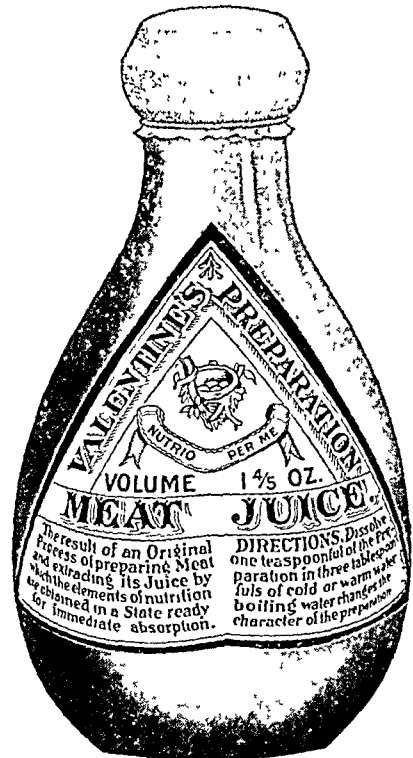
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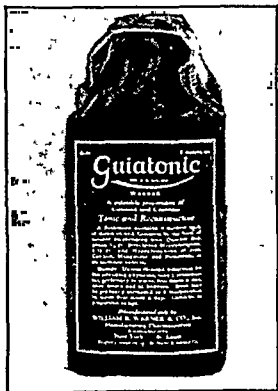
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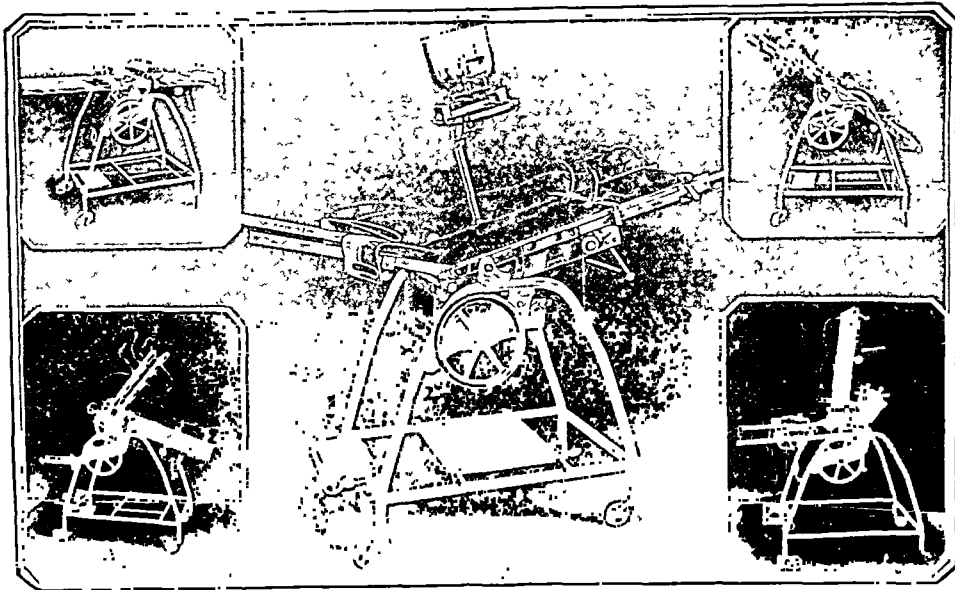
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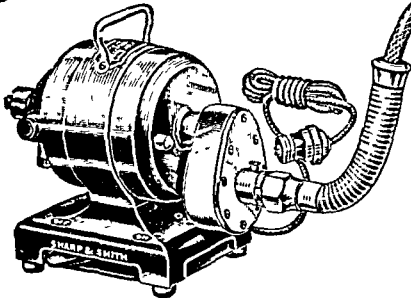
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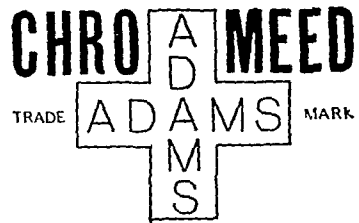
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